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CONTENTS OF PREVIOUS NUMBER

APRIL 1948. NUMBER 4

Injury of the Central Nervous System Resulting from Decompression to Simulated High Altitudes. Richard L. Masland, M.D., Philadelphia.

Clinical Aspects of Cerebral Cysticercosis. S. Obrador, M.D., Madrid, Spain.

Modification by Curare of Circulatory Changes During Electrically Induced Convulsions in Man: A Note on *d*-Tubocurarine. M. D. Altschule, M.D., and K. J. Tillotson, M.D., Waverley, Mass.

Fall in Plasma Protein Level Associated with Rapid Gain in Weight During Course of Electroshock Therapy. M. D. Altschule, M.D.; J. E. Cline, M.D., and K. J. Tillotson, M.D., Waverley, Mass.

Reflex Studies in Electroshock Treatments. Nathan Savitsky, M.D., and William Karliner, M.D., New York.

Modification of Meningeal Signs by Concomitant Hemiparesis. Melvin W. Thorner, M.D., D.Sc., San Antonio, Texas.

Effect of Optic and Acoustic Stimuli on the Cortex and Hypothalamus Under Conditions of Picrotoxin Convulsions. E. Gellhorn, M.D., Ph.D., and H. M. Ballin, Minneapolis.

Sturge-Weber Syndrome. Archibald D. McCoy, M.D., and Harold C. Voris, M.D., Chicago.

Follow-Up Study of Men with Penetrating Injury to the Brain. John A. Aita, M.D., Ph.D., Omaha.

Sleep-Electroshock Therapy of the Psychoses. H. S. Rubinstein, M.D., Ph.D., Baltimore.

Case Reports:

Erosion of the Anterior Clinoid Processes Simulating That Due to Intrasellar Tumor: A Diagnostic Pitfall. Arthur Ecker, M.D., Syracuse, N. Y.

Effect of Albumin Solution on Cerebrospinal Fluid Pressure. Frederick A. Fender, M.D., and Alexander S. MacKenzie, M.D., San Francisco.

News and Comment.

Obituaries:

Abraham Arden Brill, M.D.

Society Transactions:

New York Academy of Medicine, Section of Neurology and Psychiatry, and the New York Neurological Society.

Philadelphia Neurological Society.

Chicago Neurological Society.

Book Reviews.

Archives of Neurology and Psychiatry

VOLUME 59 MAY 1948 NUMBER 5

COPYRIGHT, 1948, BY THE AMERICAN MEDICAL ASSOCIATION

ARTERIOVENOUS ANEURYSMS OF THE BRAIN

Their Diagnosis and Treatment

HERBERT OLIVECRONA, M.D.

AND

JOHANNES RIIVES, M.D.

STOCKHOLM, SWEDEN

CLASSIFICATION

IN A MONOGRAPH published in 1936 by Bergstrand, Olivecrona and Tönnis,[1] the following classification of the angiomatous malformations and tumors was used by Bergstrand, who wrote the chapter on pathology:

1. Angioma cavernosum
2. Angioma racemosum
   (a) Telangiectasia
   (b) Sturge-Weber disease
   (c) Angioma racemosum arteriale
   (d) Angioma racemosum venosum
   (e) Aneurysma arteriovenosum
3. Angioblastoma
4. Angioglioma

The angiomatous malformations of the brain are therefore divided into two main groups, the cavernous and the racemose angiomas. The difference between these two groups is that in the racemose angiomas the blood vessels in the malformation are separated by parenchyma and in the cavernous angiomas they are not.

Among the racemose angiomas, the telangiectases and Sturge-Weber disease (nevoid amentia) are malformations of capillaries or precapillary blood vessels and are well defined anatomic and clinical entities. This cannot be said of the last three types of the racemose angiomas. In the arterial racemose angiomas, both feeding and draining vessels should theoretically be arteries. Such a lesion might exist, but no convincing case has yet been published, and among the cases described in the literature under this head in which the available facts are sufficient for the formation of an independent opinion, the draining vessels have always proved to have the anatomic character of veins. Probably all the arterial racemose angiomas described in the literature under this name are in reality arteriovenous aneurysms.

From the Neurosurgical Clinic, Serafimerlasarettet: Director, Prof. H. Olivecrona.

1. Bergstrand, H.; Olivecrona, H., and Tönnis, W.: Fefässmissbildungen und Gefässgeschwülste des Gehirns, Leipzig, Georg Thieme, 1936.

Venous racemose angiomas are rare, but a few authentic cases of this type have been described in the brain and some in the spinal cord. In these angiomas both the feeding and the draining vessels are veins. As pointed out by Cushing and Bailey,[2] most venous racemose angiomas are closely related to the arteriovenous aneurysms, and Bergstrand and associates[1] stated that they are anatomically indistinguishable from the latter. The difference is physiologic and depends on the extent of the arterial supply. If this is sufficient to give the blood in the malformation an arterial coloring the lesion should be classified as an arteriovenous aneurysm; but the differentiation can be made only in vivo; otherwise the term venous racemose angiomas may be used. Most cases described in the literature as instances of venous racemose angiomas in which observations on autopsy specimens alone are available, probably belong to the arteriovenous aneurysms.

The arteriovenous aneurysms are by far the commonest and most clinically important racemose malformations of the blood vessels of the brain. In this type the capillary net belonging to an artery, or to a group of arteries, is missing, and the arterial blood empties directly into veins. This communication between the arterial and the venous system occurs in two forms. In one there is a direct communication between an artery and a vein. In the brain this form is rare, the best known example being the case of Dandy.[3] All our cases belong to the second group, in which a tangle of abnormal blood vessels, of varying caliber, is interposed between the arterial and the venous system. The veins draining this tangle of blood vessels are dilated and pulsating and contain arterial blood. The feeding arteries are hypertrophic and dilated. The tangle of blood vessels interposed between arteries and veins may have the anatomic character of arteries or veins; most of the vessels, however, are undifferentiated, and of a pathologic structure.

CLINICAL MATERIAL

The arteriovenous aneurysms of this clinic were published in 1936 in the monograph of Bergstrand, Olivecrona and Tönnis.[1] Tönnis' chapter on the arteriovenous aneurysms was based on 22 cases, 18 of which came from the neurosurgical clinic in Stockholm and 4 from the neurosurgical clinic in Würzburg, Germany. During the period from July 1, 1935, to Dec. 31, 1946, 42 additional cases have occurred at the neurosurgical clinic in Stockholm, and it is with the experience gained in these cases that the present paper is mainly concerned. Reference to the ma-

2. Cushing, H., and Bailey, P.: Tumors Arising from the Bloodvessels of the Brain, Springfield, Ill., Charles C Thomas, Publisher, 1928.

3. Dandy, W. E.: Arteriovenous Aneurysm of the Brain, Arch. Surg. **17**:190 (Aug.) 1928.

terial already published will be made only in so far as is necessary, but since copies of the monograph can no longer be obtained we have been more liberal in this respect than would otherwise have been necessary.

FREQUENCY

The arteriovenous aneurysms of the brain are comparatively rare lesions. In the opinion of Dandy,[3] they occur in about 1 per cent of a series of tumors of the brain. Cushing and Bailey[2] noted 9 cases in a series of 1,522 verified cases of cerebral tumor. In the monograph of 1936,[1] 18 arteriovenous aneurysms in a series of 941 verified tumors of the brain were described. Up to Aug. 31, 1946, 3,206 cases of verified tumors of the brain had been observed in this clinic. Of these, 60 were cases of arteriovenous aneurysm, 54 of which were verified by arteriography, 4 by direct inspection, 1 at autopsy only and 1 by histologic examination of the extirpated aneurysm alone. One of the cases belonging in the earlier series (case 9[1]) is included in the present series, as the patient was readmitted to the clinic and the aneurysm was removed ten years after the first arteriographic examination and exploration.

In our series, slightly more than twice as large as the one reported by Cushing and Bailey,[2] the incidence of arteriovenous aneurysms is approximately seven times as great as that in their series. It is obvious that

TABLE 1.—*Age of Onset of Arteriovenous Aneurysms in Three Series*

Age of Onset	Dandy (1938), %	Bergstrand, and Tonnis Olivecrona (1936), %	Own Material (1946), %
1-10	38.9	9.5	9.17
11-20	16.7	18.1	34.2
21-30	—	36.3	34.2
31-40	16.7	18.1	12.5
41-50	27.7	18.1	9.7

the introduction and frequent use of arteriography in our clinic since 1932 are responsible for this tremendous increase in the diagnosis of arteriovenous aneurysm.

SEX AND AGE INCIDENCE

It is noteworthy that arteriovenous aneurysms occur with much greater frequency in males than in females. In the 9 cases reported by Cushing and Bailey,[2] 7 of the patients were males and 2 females, and Dandy's 8 patients were all males. Of the 22 cases of arteriovenous aneurysm reported by Bergstrand, Olivecrona and Tönnis,[1] 15 were of males and 7 of females. Of 35 other cases recently reported in the literature, 25 were of males and 10 of females, and in our present series 29 of

the patients were males and 13 females. The arteriovenous aneurysms, therefore, are more than twice as common in males as in females.

Since the arteriovenous aneurysms are congenital malformations, the first symptoms might be expected to occur at an early age. This, however, is not the case, and the appearance of symptoms before the age of 10 years is comparatively rare. The earliest onset of symptoms recorded in the literature was in a case described by Ray[4] in a child 2½ years old, and the majority of patients have their first symptoms during the second or third decade of life. In table 1 is recorded the age of onset in Dandy's material[3] (his own cases and the series of cases he collected from the literature up to 1928), the series of Bergstrand, Olivecrona and Tönnis (1936) and our own material (1946).

Since the material presented in table 1 is small and the figures show considerable variation, no great weight can be attached to them; but a certain predominance of cases with onset of symptoms during the second and third decades can be discerned. A similar tendency has also been observed in cases of arteriovenous aneurysms of the extremities (Körte,[5] Coenen,[6] Sonntag[7] and Wagner[8]. The reason that a lesion present at birth does not in the majority of cases give rise to symptoms until decades later is unknown. To some extent this may be explained by the undoubted progressive nature of the arteriovenous aneurysm, the lesion being comparatively small at birth and only gradually attaining a size capable of affecting the circulation of blood in the brain sufficiently to give rise to symptoms. Another fact to be considered in this connection is the occurrence of secondary changes in the lesion, such as rupture of blood vessels and thrombosis, which may conceivably require some time to develop. In many cases, however, it remains unknown why a lesion which must have been large for years before onset of the first symptoms does not manifest itself earlier.

LOCALIZATION

In the earlier cases of arteriovenous aneurysm reported in the literature the lesion was located predominantly, if not exclusively, in the distribution of the external carôtid artery. In cases of involvement of this

4. Ray, B. S.: Cerebral Arterio-Venous Aneurysms, Surg., Gynec. & Obst. **73**:615, 1941.

5. Körte, W.: Beitrag zur Lehre vom Angioma arteriale racemosum, Deutsche Ztschr. f. Chir. **13**:24, 1880.

6. Coenen, H.: Die Geschwülste, in Kirschner, M., and Nordmann, O.: Die Chirurgie, Urban & Schwarzenberg, Berlin, 1930, vol. 2, pt. 1, p. 74.

7. Sonntag, E.: Das Rankenangiom sowie die genuine diffuse Phlebarterieektasie und Phlebektasie, Ergebn. d. Chir. u. Orthop. **11**:99, 1919.

8. Wagner, A.: Ueber das arterielle Ranken-Angiom an den oberen Extremitäten, Beitr. z. klin. Chir. **11**:49,1894.

TABLE 2—*Clinical Data of 43 Cases of Arteriovenous Aneurysm of the Brain (July 1, 1935 to Dec. 31, 1940)*

Case No.	Sex	Age, Yr.	Length of History	First Symptom	Previous Hemorrhage	Epilepsy	Positive Neurologic Symptoms	Localization	Bruit	Treatment	Result
1	F	29	2 wk.	Intracerebral hemorrhage with hemiplegia	—	—	Right hemihypestesia; aphasia; semicoma	Left middle cerebral artery	—	Removal of clot and small aneurysm	Patient well; full earning capacity
2	M	31	2 yr.	Hemiplegia	Intracerebral hemorrhage; hemiplegia	Jacksonian fits began 1 yr. later	Right hemiplegia; some aphasia; moderate mental deterioration	Left middle and anterior cerebral arteries	—	Ligature of accessible arteries	Condition unchanged
3	M	51	10 yr.	Epilepsy	Subarachnoid hemorrhage 2 mo. before admission	+	Mental deterioration; hallucination; poor vision	Right middle cerebral and posterior large contribution from external carotid artery	+	Exploration; ligature of accessible arteries	Death 24 hrs. after operation; cardiac failure, acute anemia
4	M	28	20 yr.	Left-sided hemiplegia	Intracerebral hemorrhage	+	Left hemiplegia; considerable mental deterioration	Right middle cerebral artery	—	Aneurysm removed	No improvement; epileptic dementia
5	F	27	1½ yr.	Jacksonian fits	Intracerebral hemorrhage with right hemiplegia 5 mo. before admission	+	Somnolence; right hemiplegia; slight choking of both disks	Left middle cerebral artery	—	Aneurysm removed	Much improved for 5 yrs. partially able to work, then complete hemiplegia and aphasia; total invalidism
6	M	22	2 yr.	Subarachnoid hemorrhage	3 attacks of subarachnoid hemorrhage	—	Slight weakness of left side of face	Right anterior cerebral artery	—	Ligature of accessible arteries	Two attacks of subarachnoid hemorrhage; death 3 yrs. after operation of scarlet fever
7	M	19	12 yr.	Attacks of headache with scotoma		Jacksonian fits for 8 yrs.	Poor memory	Left middle cerebral artery	—	Aneurysm removed	Well; able to work; once a year epileptic fit
8	F	27	11 yr.	Epilepsy	Attack of subarachnoid hemorrhage 6 yr. before admission; 2nd attack 2 mo. before admission	+	Some difficulty of micturition; incipient choked disk	Left anterior cerebral artery	—	Aneurysm removed	Death 5 wk. after operation of meningitis due to open frontal sinus
9	M	28	4 yr.	Jacksonian fits	—	+	Right hemiparesis; incipient choked disk	Left middle cerebral artery	+	Ligature of left internal carotid artery	No improvement; progressive right hemiparesis; bruit persisted in 1945

TABLE 2—*Clinical Data of 43 Cases of Arteriovenous Aneurysm of the Brain (July 1, 1935 to Dec. 31, 1946)—Continued*

Case No.	Sex	Age, Yr.	Length of History	First Symptom	Previous Hemorrhage	Epilepsy	Neurologic Symptoms	Localization	Bruit	Treatment	Result
10	M	45	11 yr.	Epilepsy	Subarachnoid hemorrhage 4 mo. before admission; second subarachnoid hemorrhage ½ year later	+	—	Right middle cerebral artery	+	Removal of aneurysm	Patient able to work; slight weakness in left hand; 3 epileptic fits in 5 yr.
11	F	35	4 yr.	Hemorrhage with right hemiplegia	+	+	Right hemiparesis and hemiphypesthesia; some mental impairment	Left middle cerebral artery	—	Removal of aneurysm	Patient able to work; moderate hemiparesis persists
12	M	24	7 yr.	Jacksonian fits	—	+	Electroencephalogram negative; slight weakness of left hand and face	Right middle cerebral artery	—	Removal of aneurysm	Slight paresis of left hand; 2 jacksonian fits in 7 yr.; patient able to work
13	F	40	15 yr.	Increasing vascularity of scalp	—	—	Blurring of disk margins	Left anterior cerebral; left external carotid	+	Attempted removal of aneurysm	Patient died on table from hemorrhage
14	F	32	9 yr.	Epilepsy	—	+	Electroencephalogram negative	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
15	M	37	3 yr.	Epilepsy	—	+	Electroencephalogram negative; episodes of psychosis	Left middle cerebral	—	Ligature of left internal carotid artery	Hemiplegia and aphasia; total invalidism
16	M	15	9 yr.	Intracerebral hemorrhage	+	+	—	Right anterior cerebral artery	—	Removal of aneurysm	Patient well
17	M	30	10 yr.	Subarachnoid hemorrhage	+	+	—	Left middle cerebral artery	+	Ligature of left carotid artery; after 24 hrs. beginning hemiplegia; ligature removed	Hemiparesis and aphasia gradually disappeared, though not completely; patient died by suicide 2 yr. later
18	M	20	1 yr.	Epilepsy	—	+	Slight left hemiparesis; poor memory; mental deterioration	Right middle cerebral artery	—	Ligature of right internal carotid artery; 4 days later left hemiplegia	Patient invalid because of hemiparesis and epilepsy

TABLE 2—*Clinical Data of 43 Cases of Arteriovenous Aneurysm of the Brain (July 1, 1935 to Dec. 31, 1946)*

Case No.	Sex	Age, Yr.	Length of History	First Symptom	Previous Hemorrhage	Epilepsy	Positive Neurologic Symptoms	Localization	Bruit	Treatment	Result
19	F	40	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
20	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
21	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
22	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
23	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
24	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
25	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
26	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
27	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
28	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
29	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
30	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
31	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
32	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
33	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
34	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
35	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
36	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
37	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
38	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
39	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
40	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
41	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
42	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation
43	F	30	1 yr.	Epilepsy	—	+	—	Left middle cerebral artery	—	Ligature of left internal carotid artery	Patient well; occasional epileptic fits of same frequency as before operation

18	M	20	1 yr.	Epilepsy	—	+	Slight left hemiparesis; poor memory; mental deterioration	Right middle cerebral artery	—	Ligature of right internal carotid artery; 4 days later left hemiplegia	Patient invalid because of hemiparesis and epilepsy

Table 2.—*Clinical Data of 43 Cases of Arteriocerebral Aneurysm of the Brain (July 1, 1933 to Dec. 31, 1936)*

Case No.	Sex	Age, Yr.	Length of History	First Symptom	Previous Hemorrhage	Epilepsy	Positive Neurologic Symptoms	Localization	Bruit	Treatment	Result
19	M	42	10 yr.	Epileptic fits	—	+	—	Right external cartoid artery; right anterior cerebral artery	+	—	Patient well except for occasional epileptic fits
20	M	42	2 wk.	Intracerebral hemorrhage with hemiplegia	—	Fits appeared 1 yr. after operation	Left hemiplegia; incipient choked disk	Right anterior and middle cerebral arteries	—	Removal of clot and aneurysm	Occasional epileptic fits; patient able to work; moderate hemiparesis persisting
21	M	58	13 yr.	Epilepsy	—	+	Left hemiparesis; left hemianesthesia; mental deterioration	Right middle and posterior cerebral artery	—	Removal of aneurysm	No improvement; occasional epileptic fits; psychosis; death from cirrhosis of the liver 3 yr. after operation
22	F	24	2 yr.	Subarachnoid hemorrhage with right hemiplegia	+	Sensorial; jacksonian fits	Slight right hemihypesthesia; right-sided astereognosia	Left middle cerebral artery	—	Removal of aneurysm	Patient well and able to work but slight right hemihypesthesia persists
23	M	22	8 yr.	Epilepsy	—	+	Right hemiparesis, slight mental impairment	Left anterior cerebral artery	—	Removal of aneurysm	Patient unable to work because of epileptic dementia; 5 or 6 epileptic fits a year
24	M	21	8 yr.	Subarachnoid hemorrhage with right hemiparesis	+	+	Slight right-sided facial weakness; memory impaired	Right middle cerebral artery	—	Removal of aneurysm	Slight paresis of right hand; patient able to work; no fits
25	M	24	1½ yr.	Epilepsy	—	+	—	Right middle cerebral artery	—	Removal of aneurysm; postoperative clot	Patient partially able to work; moderate left hemiparesis and hemihypesthesia; no fits
26	F	31	2 yr.	Subarachnoid hemorrhage	Two attacks of subarachnoid hemorrhage	—	Mentality considerably deteriorated	Left middle cerebral artery	—	Exploration; aneurysm not visible on surface of brain	Patient unable to work

TABLE 2—*Clinical Data of 43 Cases of Arteriovenous Aneurysm of the Brain (July 1, 1935 to Dec. 31, 1946)—Continued*

Case No.	Sex	Age, Yr.	Length of History	First Symptom	Previous Hemorrhage	Epilepsy	Positive Neurologic Symptoms	Localization	Bruit	Treatment	Result
27	F	18	1 yr.	Subarachnoid hemorrhage with left hemiparesis	+	+	Left-sided facial weakness; slight increase in tendon reflexes on left side, choked disk.	Right middle cerebral artery	—	Removal of aneurysm; postoperative clot	Patient well; no fits; full earning capacity
28	M	13	1 wk.	Subarachnoid hemorrhage	+	—	Incipient choked disk; somnolence	Right anterior cerebral artery	—	Ligature of right internal carotid artery	Patient well 2 yr. later
29	F	17	2 yr.	Subarachnoid hemorrhage with right homonymous hemianopsia	+	+	—	Left middle cerebral artery	—	—	Occasional epilectic fits; patient otherwise well; partially able to work
30	F	30	2 mo.	Subarachnoid hemorrhage with paresis of left arm	+	+	Slight left hemiparesis	Right anterior cerebral artery	—	—	Patient well 1 yr. after admission
31	M	31	16 yr.	Epilepsy	—	+	Left hemiparesis; left hemihypesthesia; astereognosia	Right middle cerebral artery	—	Removal of aneurysm	Moderate left hemiparesis after 2 yr.; patient partially able to work; no fits
32	M	16	4 yr.	Sensorian jacksonian fits	Intracerebral hemorrhage a few days before admission	+	Complete left hemiplegia; left hemihypesthesia; choked disk	Right middle cerebral artery	—	Removal of aneurysm	Slight weakness in left arm; no fits; patient able to work
33	M	26	2 yr.	Epileptic fits	—	+	—	Left middle cerebral artery	—	Removal of aneurysm	Patient partially able to work; no fits but occasional attacks of headache with sensation of epileptic fits
34	M	43	6 yr.	Headache	—	—	Very advanced mental deterioration; left hemiparesis; complete astereognosia of left hand	Right middle and anterior cerebral arteries	—	Exploration on diagnosis of parasagittal meningioma	Died 6 wk. after operation
35	F	39	13 yr.	Subarachnoid hemorrhage; right hemiparesis	+	+	Slight right hemiparesis; memory much impaired; positive electroencephalogram	Left middle cerebral artery	—	Removal of aneurysm	Moderate hemiparesis; no fits; partially able to work

TABLE 2.—*Clinical Data of 43 Cases of Arteriovenous Aneurysm of the Brain (July 1, 1935 to Dec. 31, 1946)*

Case No.	Sex	Age, Yr.	Length of History	First Symptom	Previous Hemorrhage	Epilepsy	Positive Neurologic Symptoms	Localization	Bruit	Treatment	Result
36	M	25	2 yr.	Subarachnoid hemorrhage	+	+	Slight sensory aphasia	Left middle cerebral artery	+	Removal of aneurysm; postoperative clot; removal of clot	Patient died day after operation
37	M	42	1 yr.	Attacks of scotoma	One attack of subarachnoid hemorrhage	—	Slight blurring of disks	Right posterior cerebral artery	—	Removal of aneurysm	Patient well
38	F	47	3 yr.	Blowing noise in right ear	—	—	Increased pulsation and dilation of external carotid artery on right side	Right external carotid artery; posterior cerebral artery	+	—	Condition unchanged
39	M	13	8 yr.	Intracerebral hemorrhage with right hemiparesis	+	—	Arteriovenous aneurysm in fundus of left eye; right hemiparesis, right hypesthesia	Left middle cerebral artery	—	—	Condition unchanged
40	M	23	4 yr.	Epilepsy	—	+	—	Left middle cerebral artery	+	—	Occasional epileptic fits
41	M	19	4 yr.	Subarachnoid hemorrhage	Two attacks in 1945 with left hemiplegia	—	Left hemiparesis; slight blurring of margins of disk	Right anterior and middle cerebral artery	—	Removal of aneurysm	Improvement in paresis 6 mo. later
42	M	39	3 yr.	Trigeminal neuralgia; attacks of dizziness	—	—	—	Right vertebral artery	—	Tractotomy	Patient well and able to work 1½ yr.; then sudden death from hemorrhage
43	M	50	4 yr.	Attacks of dizziness; facial tic	—	—	Slight choked disk; some weakness of left abducens; Bruns' nystagmus; some cerebellar disturbance of gait	Left vertebral artery	—	Exploration	Condition unchanged

artery, large tangles of dilated, pulsating blood vessels are seen, and can be palpated, in the scalp. It has been our experience that in these cases branches of the internal carotid artery are equally involved and that one must be prepared to find the tangle of abnormal blood vessels, through which the direct communication between the arterial and the venous system occurs, actually located in the brain. In case 2 of the earlier se-

FIG. 1 (case 13).—Arteriovenous aneurysm in the left frontal lobe with dominant contribution from the external carotid artery.

ries[1] (page 106) the lesion was confined entirely to branches of the middle meningeal and occipital arteries, but in case 1 of the same series (page 104) an aneurysm in the dura emptied largely through dilated and arterialized veins in the brain.

In the present series there were 5 cases in which branches of the external carotid artery were greatly dilated. In 2 of these cases no cerebral symptoms were present, and the impression was gained that the lesion was situated exclusively in the distribution of the external carotid artery. In the first case (13), that of a woman aged 44, a tangle of di-

lated and pulsating blood vessels had been observed for more than fifteen years, with pulsations and a systolic murmur as the only symptoms (fig. 1 and 2 *A* and *B*). An arteriogram of the external carotid artery showed an arteriovenous aneurysm fed by the enormously dilated middle

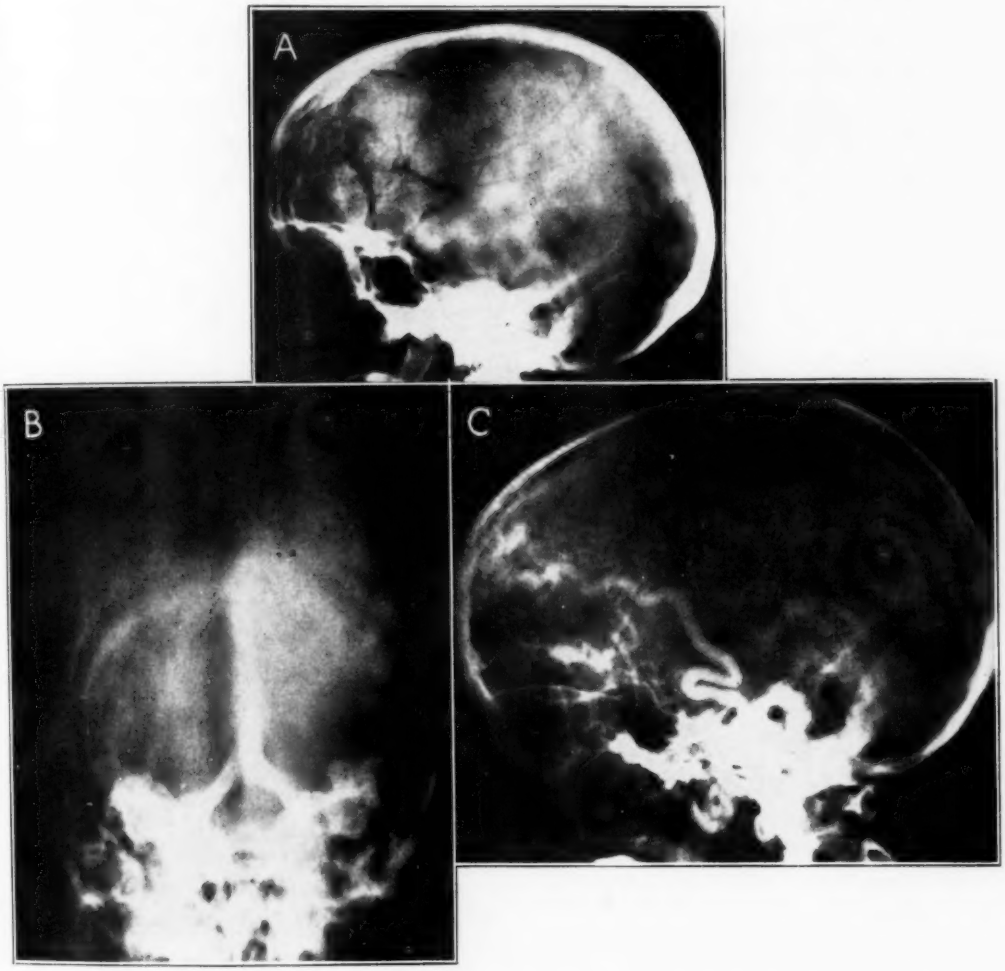

FIG. 2 (case 13).—*A*, increased vascularity of the skull. The middle meningeal artery is very much enlarged. *B*, dilatation of the sulcus for the longitudinal sinus. *C*, arteriogram of the left external carotid artery. The left middle meningeal artery is enormously dilated.

meningeal artery (fig. 2*C*). At operation the lesion was observed to extend deep into the frontal lobe, and the patient died of an uncontrollable hemorrhage. Autopsy revealed a typical wedge-shaped aneurysm, involving a considerable portion of the left frontal lobe (fig. 3). In this case the mistake was in limiting the arteriographic examination to the

external carotid artery. If an arteriogram of the internal carotid artery had been made, the true nature of the lesion would have been recognized and the operation would probably not have been attempted. It should be borne in mind that operation for arteriovenous aneurysm is particularly hazardous when both the internal and the external carotid artery are involved because of the difficulties of exposure due to communications between hypertrophic branches of the middle meningeal artery and the aneurysm.

In the second case (38) with involvement of branches of the external carotid artery, the symptoms were also referable exclusively to the hypertrophy and dilatation of these branches. Arteriograms made both

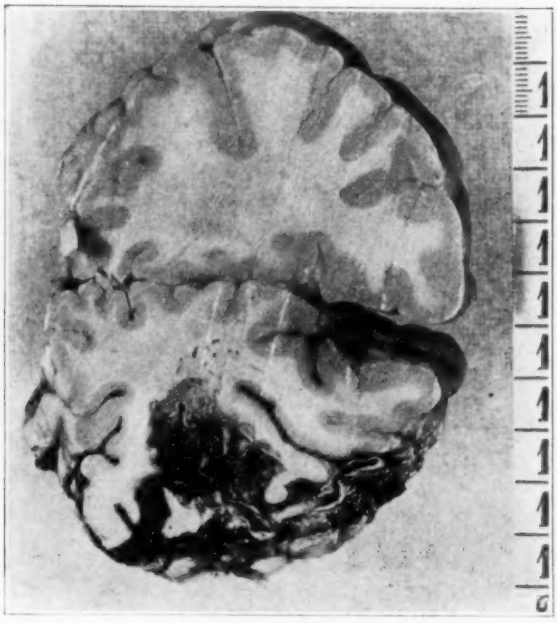

FIG. 3 (case 13).—Autopsy specimen, showing large wedge-shaped arteriovenous aneurysm, extending deep into the left frontal lobe.

of the external and of the internal carotid artery showed that the lesion extended deep into the temporal lobe. In view of the comparatively slight inconvenience caused by the aneurysm and the serious hazards to be expected in removing the lesion, operation was not advised.

In addition to cases in which branches of the external carotid artery are actively engaged as feeding arteries to the aneurysm, it is not unusual to observe hypertrophy and dilatation of the extracranial and meningeal arteries. In lesions of long standing this hypertrophy may be prominent, although never reaching the degree seen in the preceding cases.

Of the remaining 38 cases, the aneurysm was confined to branches of the internal carotid artery in 36 cases and to the vertebral artery in 2 cases. In 28 cases the feeding arteries came exclusively or predominantly from the middle cerebral artery, although in many cases some branches from the anterior cerebral, or occasionally from the posterior cerebral, artery were contributory. In 7 cases the anterior cerebral artery was exclusively or predominantly involved, and in 3 cases the anterior and the middle cerebral artery contributed to about the same extent.

On the surface of the brain as exposed at operation it is usually possible to see only a few of the arteries entering the lesion. These vessels usually disappear below the cortex at some distance from the tangle of

FIG. 4 (case 39).—Arteriovenous aneurysm of the eyeground of the left eye.

blood vessels which constitutes the bulk of the lesion, and part of which usually reaches the surface of the brain. Before entering the aneurysm, the larger arteries break up into numerous small, thin-walled blood vessels and enter the lesion at a deeper level. This gives rise to the characteristic wedge-shaped appearance of the eneurysm, which frequently reaches down to the immediate vicinity of the ventricle (fig. 3). Once this fundamental anatomic fact is clearly grasped, it becomes obvious, as already pointed out by Cushing and Bailey,[2] that ligature of visible arteries on the surface of the brain is quite incapable of materially reducing the blood supply to the lesion, since numerous arteries coming from deeper levels remain patent.

Wyburn-Mason[9] recently described an interesting localization in which an arteriovenous aneurysm of the midbrain is combined with a similar lesion in the retina. He found this combination in 9 of his own

9. Wyburn-Mason, R.: Arteriovenous Aneurysm of Midbrain and Retina, Facial Naevi and Mental Changes, Brain **66**:163, 1943.

cases and collected from the literature 20 cases of arteriovenous aneurysm of the midbrain, in 14 of which malformations of the blood vessels of the retina were also shown. The following case belongs to this type.

CASE 39.—L. E., a boy aged 5 years, who had previously been well and had developed normally, struck his head in a fall on Oct. 25, 1938. There was no loss of consciousness, and he was able to walk home by himself. The next day weakness of the right leg developed, soon extending to the arm and face. He was admitted to the clinic on November 16, where moderate right hemiparesis and angiomatous malformation of the fundus of the left eye were observed. The true nature of the lesion was not appreciated at the time, and a subdural hematoma was suspected. Encephalograms were normal, however, and, since his hemiparesis was rapidly improving, he was discharged on December 1. His improvement continued, and during the following years he was reported to be entirely well. In 1941 the hemiparesis recurred and gradually progressed, so that

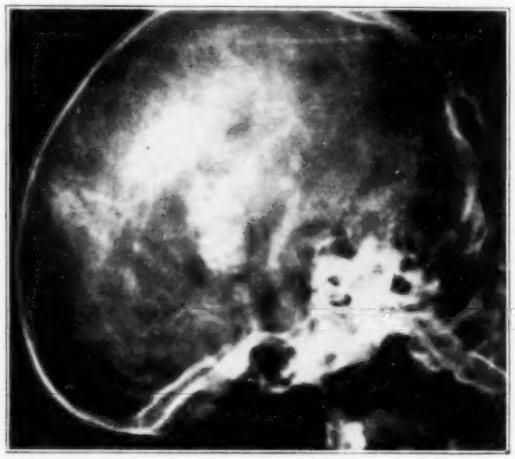

FIG. 5 (case 39).—Arteriogram of the left internal carotid artery.

his right arm became practically useless. Difficulty of speech was noted shortly before his second admission, on Sept. 12, 1946. Examination revealed right hemiparesis, pronounced in the arm and less severe in the leg and face. There was occasionally some difficulty of speech. Mental retardation was also apparent. The left eyeground presented the same picture as it had eight years earlier, and the lesion was now recognized as an arteriovenous aneurysm (fig. 4). A small calcification was visible in the roentgenograms of the head, situated slightly to the left of the midline and about 5 cm. above and a little behind the sella turcica. An arteriogram of the left side showed a small arteriovenous aneurysm at the same site as the calcification and fed by two comparatively large branches from the middle cerebral artery (fig. 5). The lesion was considered inoperable.

SIZE

At the time that the diagnosis is first made, most arteriovenous aneurysms have reached considerable dimensions. The tangle of abnormal

blood vessels, as seen in the arteriogram, is usually rather large. The caliber of arteries going to the aneurysms are several times that of normal cerebral arteries, and the entire arterial tree of the affected hemisphere is conspicuously hypertrophied. The greatest changes are seen in the veins, which are many times the size of normal cerebral veins.

Occasionally, small aneurysms are encountered. These are usually in cases in which an early hemorrhage draws attention to the lesion. A case of this type is described in the monograph[1] (case 17, page 129). Another very small aneurysm of a rather unusual type was described by one of us (H. O.) in 1927,[10] and in the present series the following case is another example of a very small aneurysm manifesting itself by an early hemorrhage.

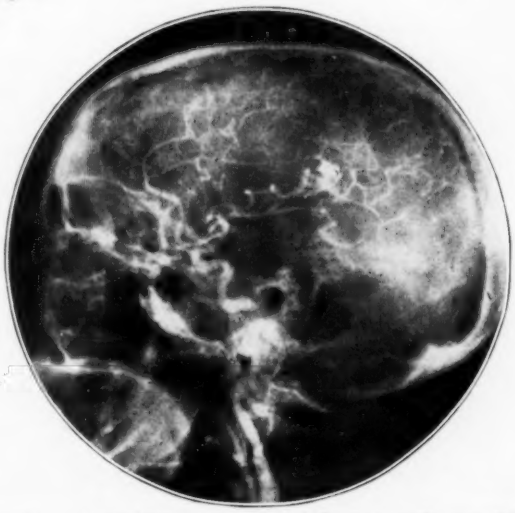

FIG. 6 (case 1).—Arteriogram of the left internal carotid artery, showing small arteriovenous aneurysm in the distribution of the middle cerebral artery.

CASE 1.—A. N., a woman aged 29, had an intracerebral hemorrhage with right hemiplegia on Nov. 12, 1935. She was admitted to the clinic on November 26. She was in the seventh month of pregnancy and was somnolent. There were complete hemiplegia of the right side, with an incipient choked disk on the same side, and aphasia.

Arteriograms showed a small arteriovenous aneurysm in the distribution of the left middle cerebral artery (fig. 6).

At operation, on November 27, a large clot was removed, together with a small aneurysm, which had been largely destroyed by the hemorrhage.

In her last letter, of August 1944, the patient reported that she was well except for slight weakness of the right hand.

At the other end of the scale, arteriovenous aneurysms of enormous proportions are encountered. The following case is an example of the huge size which is sometimes attained by lesions of this type.

10. Olivecrona, H.: Die chirurgische Behandlung der Gehirntumoren: Eine klinische Studie, Berlin, Julius Springer, 1927.

CASE 34.—G. O., a man aged 43, had always been of a peculiar mentality and had never been able to do any real work. Since the age of 6 years he had had headaches, which at times had been severe. He has been confined to bed for the last two years because of dizziness, headache and general weakness.

He was admitted to the neurosurgical clinic on Sept. 18, 1944, in poor general condition. There were bilateral moderate exophthalmos, bilateral choked disk with secondary atrophy and poor vision, left hemiparesis with astereognosis and an advanced degree of dementia.

Roentgenograms of the head showed an enormous increase in thickness of the skull, particularly in the frontoparietal region. On the right side the middle meningeal artery was enlarged, and in the superior and posterior parts of the right frontal region there was a large calcified area (fig. 7*A*).

Ventriculograms showed moderate dilatation of both lateral ventricles. The right lateral ventricle was displaced downward; the third ventricle was deformed by a calcified tumor, producing an indentation of its anterior margin.

The hyperostosis of the skull led us to suspect a parasagittal meningioma, and an exploration was done with this diagnosis in mind. The operation, however,

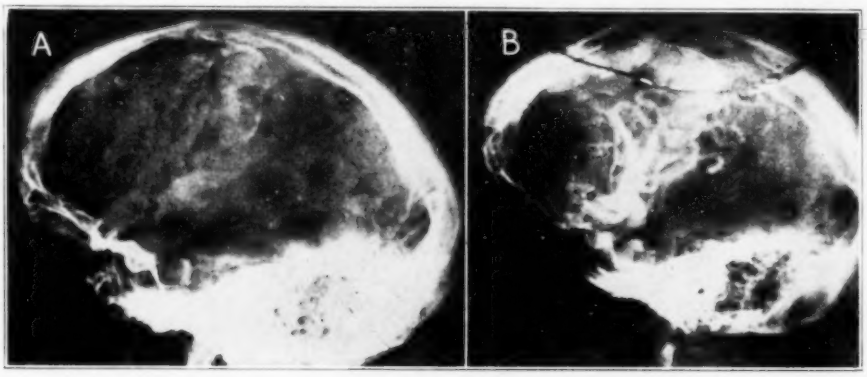

FIG. 7 (case 34).—*A*, arteriovenous aneurysm with calcification, hyperostosis of the skull and dilatation of the middle meningeal artery. *B*, arteriogram of the right internal carotid artery, disclosing large arteriovenous aneurysm.

revealed an enormous arteriovenous aneurysm. The lesion was considered inoperable, and the wound was closed.

Arteriograms confirmed (fig. 7 *B*) the presence of an enormous arteriovenous aneurysm in the distribution of the middle and anterior cerebral arteries, with extensive calcification in the walls of the vessels.

The patient left the clinic two weeks after operation and was reported to have died six weeks later.

Whether the arteriovenous aneurysm actually grows during the lifetime of the patient is as yet undetermined. It is reasonable to expect some increase in size, but the only means by which one may follow the growth of an arteriovenous aneurysm is by arteriographic examinations at long intervals, a method rarely practicable. In the following case, in which arteriographic examinations were carried out over a ten year interval, it could be shown that some increase in size had occurred during

the intervening years and that the caliber of arteries and veins feeding and draining the lesion had increased.

CASE 31.—K. B., a man aged 42, for many years had occasional attacks of headache, usually lasting for a few days. Jacksonian fits, starting in the left hand, began in 1921 and recurred a few times a year, but without loss of consciousness. In 1924, for the first time, he became unconscious during an attack, and since that time epileptic fits, beginning in the left hand and associated with loss of

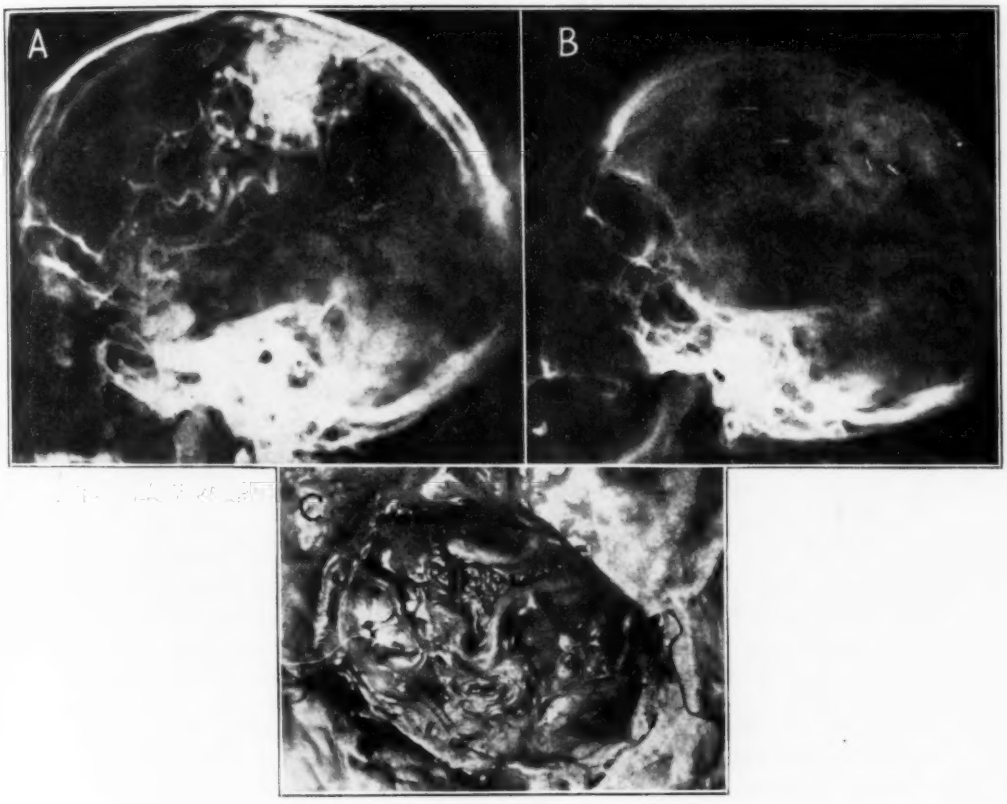

FIG. 8 (case 31).—*A*, arteriogram of the right internal carotid artery, showing large arteriovenous aneurysm in the distribution of the middle cerebral artery. *B*, arteriogram (1944), showing increase in size of the lesion since 1934. *C*, large arteriovenous aneurysm exposed at operation.

consciousness, have occurred once or twice a year. Small attacks, involving only the left hand, became frequent during 1934 and led to his first admission to the clinic, on Aug. 23, 1934. Shortly before his admission the epileptic fits began to be followed by paresis of the left arm, lasting about an hour.

Neurologic examination showed some weakness of the left arm and a slight decrease of muscle sense in the left hand.

Roentgenograms of the skull were normal.

Encephalograms showed a slight dislocation in the downward direction of the medial part of the right ventricle.

Arteriograms revealed a large arteriovenous aneurysm in the posterior part of the right frontal region, which was fed by branches of the middle cerebral artery (fig. 8*A*).

Operation 1 (Sept. 25, 1934).—An osteoplastic flap was turned down, and when the dura was opened a very large arteriovenous aneurysm was revealed. The lesion was considered inoperable and the wound was closed.

Postoperative Course.—The patient was then given roentgen therapy. He came back for examination in April 1935, after having received a series of roentgen treatments, leading to epilation of the right side of the head. There was considerable weakness of the left hand, and deep sensibility was almost entirely lost. During the following years he had occasional jacksonian fits, usually occurring two or three times a month. The condition of his left arm remained stationary for a few years, but since 1940 there had been a slow, but steady, progress of the paresis, and the paralysis extended to the left leg. He was again admitted to the clinic on Sept. 18, 1944.

There were considerable weakness of the left arm and almost complete paralysis of the hand, with some wasting and spasticity. The left leg was distinctly weaker than the right, with wasting and moderate increase in the deep reflexes.

New arteriograms showed that during the intervening years there has been a considerable increase in size of the arteriovenous aneurysm (fig. 8 *B*).

Operation 2 (Sept. 28, 1944).—After preliminary ligation of the right internal carotid artery, the old flap was again turned down. The dura was rather thick and vascular, and there were numerous adhesions between the surface of the dura and the pia. Almost the whole exposed part of the cortex was covered by a huge mass of arterialized veins and tortuous, pulsating blood vessels (fig. 8 *C*). After one or two superficial arteries to the lesion had been clipped, the pia-arachnoid at the anterior margin of the malformation was incised. The dissection was gradually deepened, being kept close to the aneurysm. The numerous entering arteries were gradually caught with mass ligatures and divided until it became possible to tilt out the whole mass, the empty veins acting as a hinge. These vessels were then divided, and the lesion was removed. After all hemorrhage from the cavity had been checked, the ligature on the carotid artery was removed, and the cavity was again inspected for bleeding points.

Subsequent Course.—There was considerable loss of blood during this long and difficult operation, and the patient was given a total of 1,000 cc. of blood before closure of the wound was begun. Immediately after operation there was complete hemiplegia of the left side, which rapidly improved, so that one month after operation the arm was in about the same condition as before, whereas the paresis of the leg was considerably more pronounced than before operation. There was also considerable dimunition of all forms of sensation in the left half of the body.

In January 1946 the patient reported that he was much improved. He could walk and was able to do light work. The condition of his arm remained about the same as before operation. There had been no epileptic attacks.

It is obvious that in this case at least the roentgen treatment which had been administered had not resulted in any shrinkage of the aneurysm.

SECONDARY CHANGES IN THE ANEURYSM

Of the secondary changes observed in arteriovenous aneurysms, hemorrhage is the most frequent and clinically important. In our material hemorrhage was observed in 22 cases, or in 50 per cent. In several cases repeated hemorrhages had occurred. In a case in which the lesion was located in the posterior fossa, the first hemorrhage terminated fatally one and a half years after the diagnosis had been made. In most cases hemorrhage was both subarachnoid and intracerebral and was usually accompanied with hemiplegia, which often was transient, although some motor defect usually remained.

Thrombosis is comparatively rare, and at operation we have never observed a fresh thrombosis in the tangle of blood vessels constituting the aneurysm or in the feeding or draining vessels. Apparently, thrombosis, when it does occur, takes place gradually and without obvious clinical symptoms. The only indication of thrombosis of part of the lesion is calcification, which appears as round shadows, representing cross sections of large blood vessels, probably veins draining the aneurysm. Case 34, described earlier, is a good example of the appearance of these calcifications, although in this case a considerable part of the calcification occurred in the walls of abnormal blood vessels without thrombosis. In case 39, also described earlier in this paper, it is doubtful whether the calcification was due to thrombosis; the appearance of the calcified area was more consistent with the view that calcification had occurred in an old clot. In the monograph[1] 2 other typical cases of thrombosis with calcification were described.

SYMPTOMS

The symptoms of arteriovenous aneurysm are comparatively well known, and little that is new has been added since the publication of the earlier work.[1] The cardinal signs are epileptic fits, subarachnoid hemorrhage and, since the majority of aneurysms are located in the territory of the middle or the anterior cerebral artery, hemiplegia. Other signs are less common but may occasionally, in combination with one or more of the preceding symptoms, be pathognomonic, particularly a systolic bruit or a calcification of typical appearance.

The first symptoms usually occur at a comparatively early age, in the majority of cases during the second or third decade. In nearly half our material the first symptom was an epileptic fit; in about one-fifth the cases cerebral hemorrhage with hemiplegia was the first symptom, and in another one-fifth the illness was initiated by a typical subarachnoid hemorrhage. In the remaining cases uncommon or uncharacteristic symptoms were the first to be noted.

It is rather characteristic that the symptoms at first occur at long intervals, during which the patient has good health, until years later the

initial symptom recurs or a new one appears. Gradually, as the illness progresses, tendency to increasing frequency and intensity of symptoms is discernible. This tendency is well illustrated by case 11, in which the patient had a nocturnal epileptic fit at the age of 16 and was then entirely well until five years later, when a subarachnoid hemorrhage occurred. From this the patient recovered and was free of symptoms for six years, when a new subarachnoid hemorrhage occurred, this time followed by epileptic fits, which recurred with increasing frequency. Arteriograms then showed an arteriovenous aneurysm in the left frontal lobe.

Epileptic fits are not only the commonest initial symptom but the most constant of all signs. Of our 43 cases, epileptic fits were absent before operation in 13. Among these, the aneurysm in 2 cases (42 and 43) was located in the posterior fossa and in 2 cases (26 and 39) in the basal ganglia. In 3 cases (13, 37 and 38) the main blood supply came from the external carotid artery, although the aneurysm itself was actually located within the brain. In 2 cases (1 and 20) the aneurysm was removed within a few days after the first symptom, cerebral hemorrhage with hemiplegia, had occurred. In 1 of these cases (20) epileptic fits developed later; in the other the patient was still well twelve years after operation. There remain 3 cases (6, 34 and 41) in which epileptic manifestations did not occur.

The epileptic fits in cases of arteriovenous aneurysm frequently have the character of jacksonian seizures, although a true hemiplegic pattern of the fit is observed only in a few cases. Spread of the discharge to the other side and early loss of consciousness are far commoner. Sensory jacksonian fits are occasionally seen, and a sensory aura followed by one-sided or generalized convulsions may also be observed. Postictal paralysis or aphasia, usually lasting only a few hours, is not common but may occasionally be encountered. Only in 4 of our 30 cases with a history of epileptic fits was a definite pattern of seizures allowing localization absent. In 3 of these cases the aneurysm was located in one frontal lobe, and in 1, in the right temporal lobe.

Petit mal was observed in 3 cases, alternating with generalized or jacksonian seizures, and in 1 case uncinate fits were seen.

The frequency of epileptic manifestations in cases of arteriovenous aneurysm is extremely variable. When these occur in the form of generalized convulsions, with or without a beginning jacksonian seizure or aura, the interval between attacks is frequently long—several months, or even years. Pure jacksonian fits usually have a much greater frequency and may periodically at least occur daily or several times a day. The general impression of the character of epileptic manifestations of arteriovenous aneurysms is that there is much more variation with regard to type and frequency of attacks than in cryptogenic or traumatic epilepsy.

Subarachnoid hemorrhage was observed in 15 cases (35 per cent) and was the initial symptom in 11 cases. Subarachnoid hemorrhage has a tendency to recur, and in 3 cases it occurred twice and in 1 case five times. The clinical picture of subarachnoid hemorrhage accompanying arteriovenous aneurysm is the same as that of subarachnoid hemorrhage from other causes.

Hemiplegia or hemiparesis, aside from the transient hemiparesis sometimes observed immediately after an epileptic fit, occurred in about one-half our cases. In 14 cases hemiplegia occurred suddenly as a result of cerebral hemorrhage; in the remaining cases the onset was gradual, being occasionally caused by thrombosis, or more frequently by the relative anemia in the distribution of the middle cerebral artery, resulting from the short circuiting of the blood through the aneurysm. Sensory and aphasic disturbances may, of course, be present.

Headache is a frequent complaint but is usually not characteristic. Occasionally, however, attacks of headache closely simulating migraine occur. Cases of this type have been described by Baruk,[11] Deutsch and Friedmann[12] and Hyland and Douglas.[13] In all these cases the aneurysm was located in the occipital lobe. In 1 of our cases (10) that of a youth aged 19, who for eight years had had jacksonian fits, attacks of typical migraine, preceded by scintillating scotoma, began to occur four years before his admission. An arteriovenous aneurysm in the parieto-occipital lobe on the medial side of the lateral ventricle was observed and removed. Attacks of scotoma and headache continued after operation, but were much less frequent.

Since the majority of patients having headache show no signs of increased intracranial pressure, some other explanation for the occurrence of headache must be sought. In our opinion, anoxemia of the brain due to shunting of the blood through the abnormal arteriovenous communication is the most likely cause. Anoxemia of the brain results in dilatation of cerebral vessels, which is known to be painful, and in the end the effect is much the same as that in expansive lesions with increased general pressure.

In the earlier literature the occurrence of a choked disk was said to be a frequent symptom. In our experience this is not so, and choked disk, as well as other signs of increased intracranial pressure, is usually

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11. Baruk, H.: Migraines d' apparence psychogenique suivies d'épilepsie jacksonienne dans un cas d'angiome cérébral, Encephale **26**:42, 1931.

12. Deutsch, L., and Friedmann, J.: Ueber ophthalmische Migräne bei Gefässmissbildung (arteriovenöse Aneurysmen), Deutsche Ztschr. f. Nervenh. **146**: 199, 1938.

13. Hyland, H. H., and Douglas, R. P.: Cerebral Angioma Arteriale: A Case in Which Migraninous Headache Was the Earliest Manifestation, Arch. Neurol. & Psychiat. **40**:1220 (Dec.) 1938.

lacking. This was pointed out in the monograph[1] in which a high degree of choked disk and a great increase in intracranial pressure were recorded only once, in a case of aneurysm in the posterior fossa. In the present series a mild degree of choked disk was observed in 11 cases, but among these there were 3 of recent subarachnoid hemorrhage. In the remaining 8 cases the choking did not exceed 2 D., and in most cases only a slight blurring of the margins of the disk was observed. In 1 case (34) there was considerable secondary atrophy, probably due to repeated subarachnoid hemorrhages.

Increased vascularity of the eyegrounds with dilatation and tortuosity of the retinal vessels has been described by Eimer and Mehlhose,[14] Dandy,[3] Cushing and Bailey,[2] Brock and Dyke,[15] Deutsch and Friedmann,[12] Northfield[16] and others. No changes of this type have been observed in our material, except in the case of an arteriovenous aneurysm of the retina already described.

Exophthalmos has been observed in cases of arteriovenous aneurysms of the brain but seems to be rare. The exophthalmos may be unilateral, and then always on the side of the lesion. Occasionally it is pulsating, as in cases described by Kreutz,[17] Schmidt[18] and Northfield.[16] We have seen bilateral exophthalmos of moderate degree in 1 case (34), mentioned earlier in this paper, and unilateral exophthalmos on the side of the lesion in another, located in this case in the right parieto-occipital region. On the whole, it is a rare symptom but may be of some value when not associated with increased intracranial pressure.

A systolic bruit was first described by Steinheil[19] in 1895 and has since been repeatedly observed and considered as one of the more important symptoms of arteriovenous aneurysm. Cushing and Bailey[2] found it in 8 of their 9 cases and were inclined to consider a bruit as one of the most frequent and important symptoms. Increasing experience has shown, however, that a systolic bruit is not as frequent as the earlier literature would lead one to think. Dandy[20] pointed out that a systolic

14. Eimer, K., and Mehlhose, K.: Das klinische Bild des zerebralen Angioma racemosum arteriale, München. med. Wchnschr. **74**:836, 1927.

15. Brock, S., and Dyke, C.: Venous and Arterio-Venous Angiomas of the Brain, Bull. Neurol. Inst. New York **2**:247, 1932.

16. Northfield, D. W. C.: Angiomatous Malformations of Brain, Guy's Hosp. Rep. **90**:149, 1940.

17. Kreutz, A.: Ueber einen Fall van Rankenaneurisma der Arteria ophtalmica dextra, Wien. med. Wchnschr. **53**:1725, 1903.

18. Schmidt, O.: Beitrag zur Klinik und chirurgischen Behandlung des Angioma arteriale racemosum, besonders des Kopfes, Beitr. z. klin. Chir. **118**:178, 1920.

19. Steinheil, S. O.: Ueber einen Fall von Varix aneurysmaticus im Bereich der Gehirngefässe, Inaug. Dissert., Würzburg, F. Fromme, 1895.

20. Dandy, W., in Lewis, D.: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc. 1932, p. 433.

bruit is sometimes absent, and in our earlier material published in the monograph[1] its presence was recorded in only 25 per cent of the cases. Röttgen[21] and Sorgo[22] frequently failed to note a systolic bruit, and in our present series this symptom was noted only in 8 of 43 cases, or in about 19 per cent. All our patients were carefully examined for the presence of a murmur, but even after the lesion had been accurately located by means of arteriograms the bruit was missed in the majority of cases. Even though, admittedly, a faint murmur may occasionally be overlooked, it is certain that a systolic bruit cannot be considered a symptom of great frequency. If present, it is of almost pathognomonic significance, although it should be borne in mind that a bruit is occasionally observed with saccular aneurysms or with very vascular meningiomas. No doubt the earlier literature contains many observations on inveterate cases, in which the frequency of a bruit might conceivably be greater. This is probably also true of increased vascularity of the scalp and bone and of secondary hypertrophy of the heart due to an increased volume of blood. As already mentioned, increased vascularity of the scalp and bony cranial structures is sometimes observed even in comparatively early cases, but hypertrophy of the heart, which has been observed with considerable frequency with arteriovenous aneurysms of the extremities, is practically never seen with arteriovenous aneurysm of the brain.

It has long been recognized that mental changes are frequent with arteriovenous aneurysms of the brain, particularly those of long standing, and the early authors, such as Emanuel[23] and Steinheil,[19] have commented on the mental deterioration of their patients. In our material mental changes were observed in about 50 per cent of cases. In about one-half this number the changes were slight, but in the other half they were frequently pronounced, and sometimes the end result of a long period of illness was an advanced state of dementia. Episodes of frank psychosis, necessitating temporary confinement in an asylum for mentally disturbed patients, was occasionally observed. Numerous epileptic fits and destruction of brain tissue through intracerebral hemorrhage are important contributory factors, but the progressive atrophy of the brain caused by short circuiting of the blood through the aneurysm is probably the most important cause of the mental changes so frequently observed in the more advanced stages of arteriovenous aneurysm of the brain.

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21. Röttgen, P.: Weitere Erfahrungen an kongenitalen arterio-venösen Aneurysmen des Schädelinnern, Zentralbl. f. Neurochir. **2**:18, 1937.

22. Sorgo, W.: Weitere Mitteilungen über Klinik and Histologie des kongenitalen arterio-venösen Aneurysmas des Gehirns, Zentralbl. f. Neurochir. **3**:64, 1938.

23. Emanuel, C.: Ein Fall von Angioma arteriale racemosum des Gehirns Deutsche Ztschr f. Nervenh. **14**:288, 1899.

Arteriovenous aneurysms of the posterior fossa are rare, and the clinical picture is not characteristic. When located in the cerebellum, as in our 2 earlier cases published in the monograph,[1] general pressure symptoms and cerebellar disturbance of gait and station are prominent features. In 1 of these cases there was, in addition, a typical trigeminal neuralgia. When the aneurysms are located in the cerebellopontile angle as in the 2 cases in our present series, the most conspicuous symptoms occurred from irritation of cranial nerves—trigeminal neuralgia and vertiginous attacks in 1 case and facial tic and tinnitus in the other. Until there has been much more experience most arteriovenous aneurysms in the posterior fossa will probably be accidentally discovered during exploration for presumed cerebellar tumor.

ROENTGENOLOGIC CHANGES OF THE SKULL

Increased vascularity of the skull was observed in 9 cases, or approximately 20 per cent of the series. This is a constant feature when the arterial supply derives wholly or in part from branches of the external carotid artery and was particularly conspicuous in case 13, in which even the sulcus caused by the longitudinal sinus was greatly enlarged. This most unusual sign of increased vascularity should have served as a warning as to what was to be expected in attacking this formidable lesion. As is to be expected, the presence of a bruit is much more frequently observed when there is a pronounced increase in the vascularity of the skull, indicating a free participation of the external carotid artery, and should as a rule indicate an arteriographic study of both the internal and the external carotid artery. Sometimes the roentgenologic picture closely resembles that of a vascular meningioma with increase in the diameter of the foramen spinosum and enlarged channel for the middle meningeal artery, and in 1 of our cases (case 34), already mentioned, the vascular changes, the increased thickness of the skull and the presence of calcification actually led to the preoperative diagnosis of a meningioma.

When the vascular supply comes entirely, or almost entirely, from the internal carotid artery, changes of the bony structures are usually lacking, even though some increase in the vascularity of the bone and the dura may be noted at operation.

Calcification is not common and was observed in 6 of our cases, or about 14 per cent. In at least 3 of these cases there was a history of intracerebral hemorrhage, and the calcification in all probability occurred in an old clot. In case 34 the walls of blood vessels showed extensive calcification, and to a lesser extent this was observed in the remaining 2 cases. There was no case of calcified thrombosis similar to that observed in case 15 in the monograph.[1]

Encephalograms were made in the majority of cases. The most frequent change noted was cerebral atrophy, either general (6 cases) or local

(11 cases). Sometimes a cavity in the brain from an old hemorrhage was filled with air, giving the appearance of porencephaly. In 13 cases the malformation was large enough to cause dislocation of the ventricular system, indicating an expansive lesion. The dislocation was always comparatively slight and was sometimes associated with atrophy of the brain, a finding that must be regarded as strong evidence of arteriovenous aneurysm.

DIAGNOSIS

A history of epileptic seizures, particularly of jacksonian type, with one or several episodes of intracranial hemorrhage, makes the diagnosis of arteriovenous aneurysm practically certain. The presence of a bruit is also pathognomonic, but a murmur is a rare symptom when the blood supply, as in the majority of instances, comes entirely from branches of the internal carotid artery.

In the absence of the aforementioned symptoms, or combination of symptoms, the diagnosis may be in doubt. A saccular aneurysm is a frequent cause of hemorrhage, subarachnoid or intracerebral, and epileptic fits may be a sequel to hemorrhage. A slowly growing fibrillary astrocytoma may be the cause of jacksonian seizures continuing for many years, and in not a few cases the fits are for a long time diagnosed as cryptogenic epilepsy. In every case in which there is reason to suspect an arteriovenous aneurysm an arteriogram should be made. During the last few years arteriograms in our clinic have been made by percutaneous injection of "per-abrodil" ("diodrast," iodopyracet U. S. P.) a procedure that appears to be innocuous and much superior to the old method of injecting "thorotrast" (a colloidal suspension of thorium dioxide) into the exposed carotid artery, a method both cumbersome and time consuming and not entirely devoid of dangers. Arteriograms, of course, always disclose the presence of an arteriovenous aneurysm. Theoretically, it is possible that an arteriovenous aneurysm deriving its blood supply entirely from branches of the posterior cerebral artery might escape detection if the injection were made in the common or the internal carotid artery. In our few cases of arteriovenous aneurysm situated in the distribution of the posterior cerebral artery there were always sufficient communications with branches of the middle cerebral artery to insure filling of the aneurysm through injection into the carotid artery. There is one source of error in connection with the interpretation of arteriograms which should be pointed out here, since it might lead to such gross errors as localizing the aneurysm in the wrong hemisphere. Such a large part of the total blood supply to the brain may pass through the aneurysm that the malformation may be filled with contrast medium injected into the contralateral carotid artery. This happened in case 10.

CASE 10.—K. L., a man aged 45, had had epileptic fits since the age of 10 years. Four months before his admission he had an acute subarachnoid hemorrhage. He was admitted to the clinic on May 9, 1939. Neurologic examination revealed nothing abnormal. Encephalograms showed slight dilatation of both ventricles, a little more pronounced on the left. Arteriograms in the left carotid artery showed a large arteriovenous aneurysm, presumably in the left parietal region (fig. 9*A*).

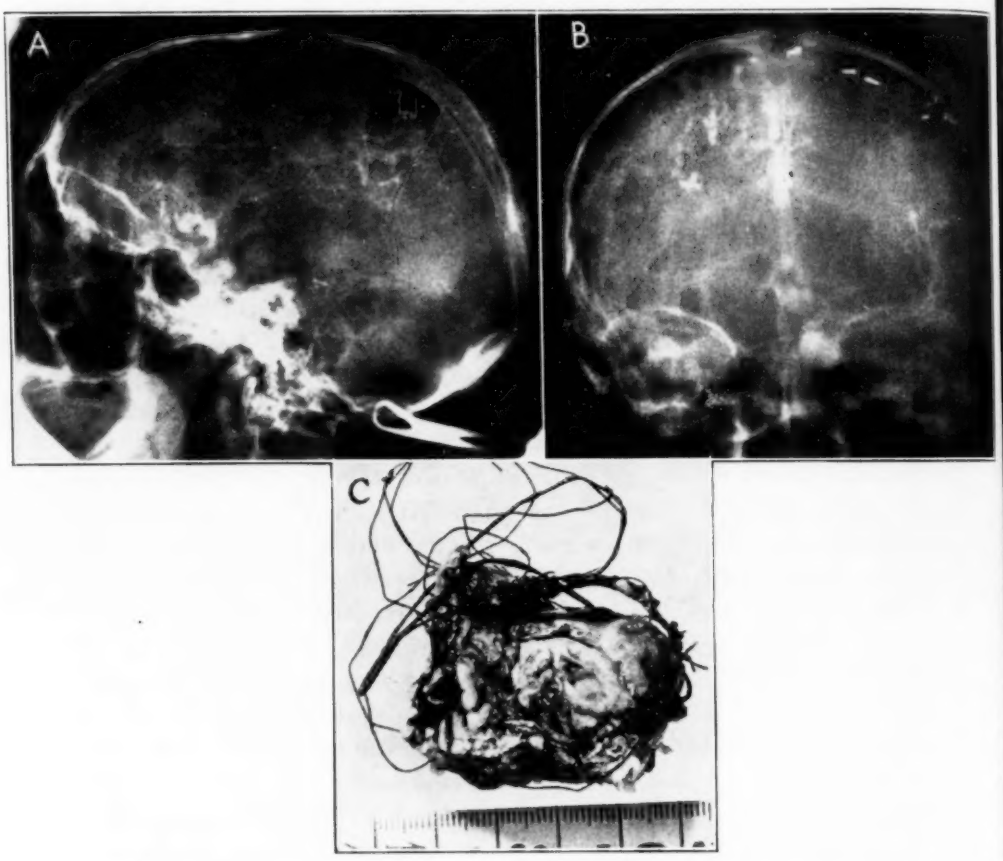

FIG. 9 (case 10).—*A*, arteriogram of the left carotid artery, showing arteriovenous aneurysm in the parietal region. The aneurysm was actually located on the right side but filled also from the left side. *B*, arteriogram of the left internal carotid artery, disclosing that the aneurysm was actually located on the right side, although it could be partially filled by injection of the left carotid artery. *C*, operation specimen.

Operation 1 (May 15, 1939).—A large osteoplastic flap was turned down over the left parietal region. The brain looked entirely normal, and no sign of an arteriovenous aneurysm was seen. The wound was closed, and the patient left the hospital two weeks later.

Postoperative Course—On November 7, a second subarachnoid hemorrhage occurred. He was readmitted to the clinic on Jan. 13, 1940. Neurologic examination revealed a normal condition. New arteriograms were made, this time with

both frontal and lateral exposures. These showed (fig. 9*B*) that the aneurysm was actually located in the right parietal region.

Normally, the circulation in the two internal carotid arteries is so nicely balanced that after injection of one carotid artery the contralateral artery is never filled. In cases of arteriovenous aneurysm, however, this occurs, as the rapid circulation through the malformation tends to suck in not only blood from the homolateral carotid artery, the normal branches of which usually show remarkably poor filling, but blood from the contralateral carotid artery as well.

Operation 2 (Feb. 19, 1940).—A large flap was turned down over the right parietal lobe. A large arteriovenous aneurysm, covering most of the exposed cortex behind the central sulcus, was observed and removed (fig. 9*C*).

Subsequent Course.—Immediately after operation there were moderate hemiparesis and hemihypesthesia of the left side. At the time of his discharge, on March 16, 1940, the hemiparesis had improved but was still rather pronounced.

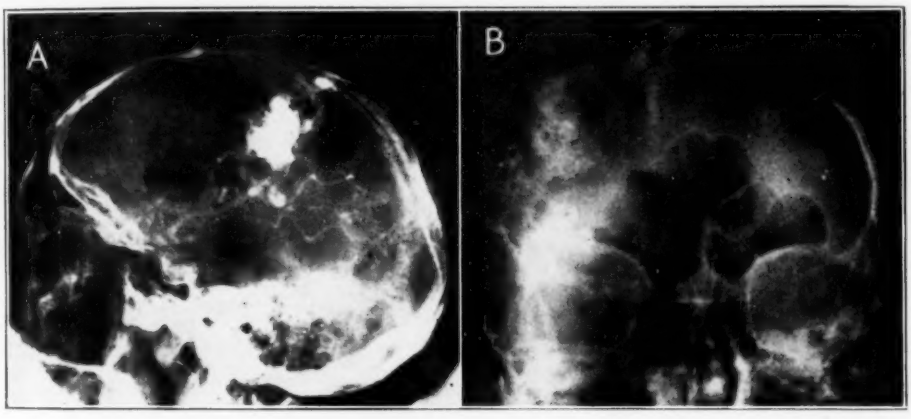

FIG. 10 (case 12).—Arteriograms in the case of a man aged 24 with a history of jacksonian fits for seven years and gradually increasing weakness of the left hand and left side of the face. *A*, large aneurysm in the distribution of the right middle cerebral artery. *B*, frontal exposure, showing the wedge-shaped aneurysm and large veins draining into the longitudinal sinus.

The patient's condition continued to improve, and in his last letter of July 1946 he reported that he was well and able to work. His only complaint was hemihypesthesia on the left side, which still persisted, and occasional epileptic fits during the first three years after operation.

If there are no definite lateralizing signs, a frontal exposure must always be made to determine which hemisphere is the site of the malformation. In addition to their diagnostic value, arteriograms are of the greatest importance in deciding the operability of the lesion and in planning the operative attack. In some cases the arteriogram shows a lesion of such dimensions that an attempt at removal would appear foolhardy. In other cases the situation of the aneurysm is such, in the basal ganglia for instance, that removal is impossible.

Before the removal of an arteriovenous aneurysm is attempted, arteriograms with both lateral and frontal exposures should be made. It is im-

portant to obtain as much information as possible as to the situation, number and caliber of the arteries supplying the aneurysm and the number and situation of the veins. If branches from the external carotid artery contribute to the blood supply to the aneurysm, separate arteriograms of the internal and external carotid arteries should be made.

TREATMENT

Not so long ago the arteriovenous aneurysms of the brain were considered hopeless lesions. Cushing and Bailey[2] recommended decompression or roentgen treatment if the diagnosis was made before exploration. Dandy stated that most of such aneurysms were inoperable and recommended decompression when pressure symptoms were present[20] except in those rare cases in which a solitary fistula between artery and vein made ligature of the abnormal connection feasible.

The first case on record in which an arteriovenous aneurysm was successfully removed in its active stage occurred in this clinic in 1932. The aneurysm was a large one in the posterior fossa, and the patient is still alive and able to work. In the monograph of 1936[1] 4 additional cases were recorded in which arteriovenous aneurysms had been successfully excised. Nevertheless, our impression of the hazards attending this operation still made us reluctant to recommend removal of the malformation except when conditions were comparatively favorable, as when the aneursym was small or was located in the distribution of the external carotid artery.

Experience with cases collected in our monograph,[1] however, had convinced us of the uselessness of partial occlusion of the arterial supply by clipping those arteries which were readily accessible. No beneficial effect was noted in any of the cases in which this method was given a trial. Since pressure symptoms on the whole are rare, we concluded that decompression was seldom advisable, and in most cases contraindicated, as the danger of hemorrhage from the aneurysm might conceivably be increased.

We were unable to find any proof that roentgen treatment might cause shrinkage of the lesion or in any way alter the spontaneous course of the illness. In figure 8*B* of the present paper are shown arteriograms after a ten year interval in a case in which roentgen treatment had been extensively employed during the first year after the first arteriogram was taken. The arteriograms show clearly that in spite of the roentgen treatment the lesion had continued to expand. Nor could any clinical improvement be noted; on the contrary, there was steady progress of the symptoms.

Ligature of the carotid artery has been fairly widely employed, but little has been published about the results. Dandy[20] expressed the belief

that there might be some justification for this procedure, and in the monograph[1] the opinion was expressed that ligature of the carotid artery and removal of the aneurysm were the only methods of treatment deserving further trial. Later experience, however, has convinced us that ligature of the carotid artery is actually much more dangerous than excision of the malformation. Of 6 cases in which this method was tried, hemiplegia resulted in 4 and became permanent in 2, in which the liga-

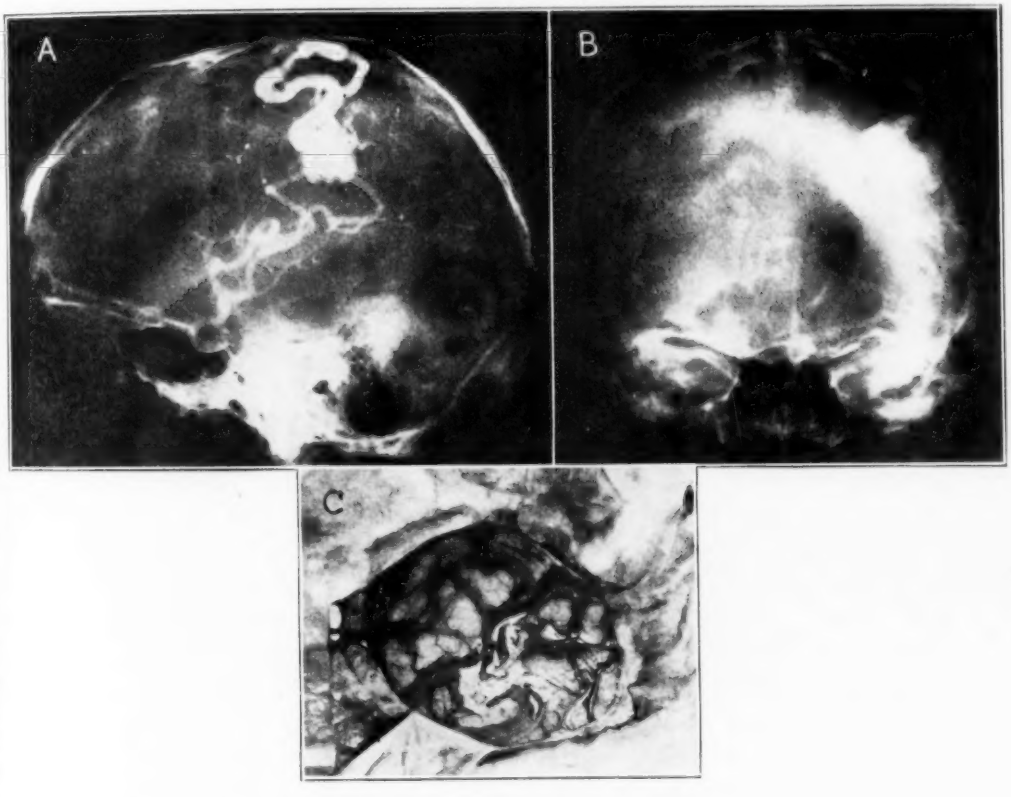

FIG. 11 (case 33).—Arteriograms of the left carotid artery in the case of a man aged 26 with a history of epileptic fits occurring several times a day for two years. *A*, arteriovenous aneurysm in the distribution of the middle cerebral artery. *B*, frontal exposure. *C*, arteriovenous aneurysm exposed at operation.

ture was not removed in time. In none of the cases in which the ligature was tolerated could it be said with any certainty that the spontaneous course of the illness was changed. The reason for the disastrous effects of ligation of the carotid artery is probably that short circuiting of the blood through the aneurysm results in severe anoxemia of the affected hemisphere when the total blood supply to the brain is suddenly diminished by tying off one carotid artery. In an earlier paper by one of us

(H. O),[24] it was also shown that the arteriovenous aneurysms of the cavernous sinus react toward ligation of the carotid artery in much the same way as the type of arteriovenous aneurysm here under discussion, and that the saccular aneurysms tolerate ligature of the carotid artery far better.

We gradually came to the conclusion that the choice lies between removing the lesion and leaving it alone. From the scant reports in the literature, it would appear that other neurosurgeons are proceeding along similar lines. In the monograph on epilepsy by Penfield and Erickson[25] are reported 2 cases in which an arteriovenous aneurysm was successfully removed; Pilcher[26] reported 3 cases and Dott[27] 7 cases, of successful removal of arteriovenous aneurysm. So far, these reports are the only one in the literature which we have been able to find.

In 24 of the present series of 43 cases of arteriovenous aneurysm, radical excision of the lesion was carried out. In the light of present experience, similar treatment should have been carried out in some of the remaining 19 cases. Instead, various procedures were tried, which as a rule were of little benefit. In 3 cases ligature of accessible arteries was done, with death from cardiac failure the day after operation in 1 case, that of a man aged 51, and no appreciable benefit in the other 2 cases. In 6 cases ligature of the carotid artery was carried out, with permanent hemiplegia in 2 cases and no benefit in the others. In 2 cases of aneurysm situated in the posterior fossa the lesion was considered inoperable. In 1 of these cases, that of a man aged 39, tractotomy for the relief of trigeminal pain was done. The patient died one and a half years later of cerebral hemorrhage, presumably originating in the aneurysm. In 2 cases very large aneurysms situated in the distribution of the middle cerebral artery were explored, in 1 case under the erroneous diagnosis of parasagittal meningioma (case 34). In both the aneurysm was considered inoperable, and 1 of the patients died six weeks after operation. In 6 cases either the symptoms did not justify such a hazardous procedure as removal of the aneurysm or the arteriogram indicated that the lesion was inoperable and it was left alone. Of the 19 patients whose aneurysms were not removed entirely, 5 have died, 1 in the clinic from the immediate effect of the operation, and the others at intervals of from six weeks to three years after operation.

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24. Olivecrona, H.: Ligature of the Carotid Artery in Intracranial Aneurysms, Acta chir. Scandinav. **91**:353, 1944.

25. Penfield, W., and Erickson, T.: Epilepsy and Cerebral Localization, Springfield, Ill., Charles C Thomas, Publisher, 1941, pp. 168 and 254.

26. Bancroft, F. W., and Pilcher, C.: Surgical Treatment of the Nervous System, Philadelphia, J. B. Lippincott Company, 1946, p. 243.

27. Dott, N.: Personal communication to the authors.

In the group of 24 patients whose aneurysms were radically removed, 3 fatalities occurred. One patient died on the table of a hemorrhage that could not be controlled; 1, of the effects of a postoperative clot, and 1, of meningitis due to accidental opening of the frontal sinus. The mortality, therefore, was 13 per cent. With the addition of the 5 cases belonging to our earlier series in which the aneurysm was removed, the mortality is reduced to 10.7 per cent.

Of the survivors, 8, or one third of the group, were practically well, with full, or nearly full, earning capacity. Another 8 patients had defects which in most instances had been present before operation, but sometimes were aggravated. These patients were partially able to work. The remaining 5 survivors were all total invalids. Of these 5 patients, 1 was well, except for a slight weakness of the right leg, for five years, when she suddenly had a series of epileptic fits with complete hemiplegia and aphasia. Probably there was a recurrence in this case. The remaining 4 patients were all total invalids before operation, because of hemiplegia, mental deterioration or severe epilepsy, but their condition was not improved by removal of the malformation.

The prognosis with respect to epileptic fits, nearly always present in cases of arteriovenous aneurysms, is of special interest. Of the 24 patients, 19, or 80 per cent, had epilepsy before operation. Eight, or one third, had no fits after operation; another third had occasional fits, perhaps once or twice a year. One of these patients had no fits before operation. He was operated on two weeks after the occurrence of his first symptom, intracerebral hemorrhage with hemiplegia. Three patients continued to have fits with the same frequency as before operation, and 1 was operated on too recently to permit any conclusion. The prognosis of epilepsy is best in the case of younger persons with a short history of epilepsy, while in cases of inveterate disease with a long history of epilepsy the outlook is poor.

When one is considering the indications for operation, one should remember that almost half our patients had a history of one or more hemorrhages with hemiplegia, which was usually transient, although some degree of motor defect generally remained. In the end, probably most, if not all, patients die of hemorrhage or are completely incapacitated. Mental deterioration is a common feature in cases of an inveterate lesion, a fact that also strengthens the argument for removal of the lesion. On the other hand, it should be realized that in some cases the condition is present for years without other symptoms than an occasional epileptic seizure. In the absence of disabling symptoms or hemorrhage, removal of the lesion should not be considered if the aneurysm is large and is situated in the distribution of the middle cerebral artery. Occasionally, even though the symptoms are comparatively mild, the arterio-

gram shows an aneurysm of such formidable dimensions that the wise course is to leave it alone, particularly when the lesion is in the left hemisphere. The poor prognosis for epilepsy in cases of an inveterate lesion should also serve as a warning against too much surgical zeal, particularly if the patient is already incapacitated from mental deterioration, hemiplegia or other complication.

OPERATIVE TECHNIC.

Control of hemorrhage is the chief difficulty in the removal of an arteriovenous aneurysm, and in spite of every precaution the loss of blood

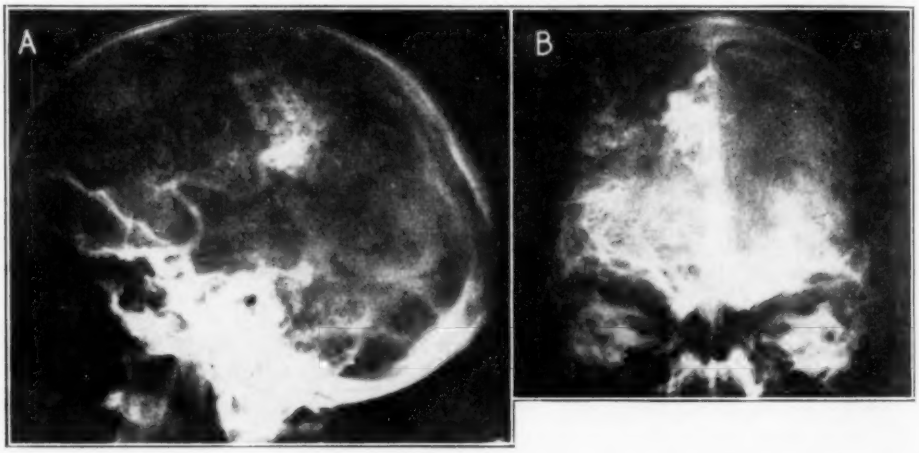

FIG. 12 (case 35).—Arteriograms of a woman aged 39 with a history of subarachnoidal hemorrhage thirteen years before, followed by right hemiparesis and epileptic fits involving the right side. *A*, arteriogram of left internal carotid artery, showing large arteriovenous aneurysm in the distribution of the middle cerebral artery. It should be noted that practically none of the normal cerebral arteries were filled, as most of the blood coming through the left carotid artery was diverted through the aneurysm. *B*, frontal exposure, showing wedge-shaped aneurysm extending deep into the sylvian fissure.

may be considerable. Intravenous infusion should be maintained during the entire procedure, and blood in sufficient quantities should be ready for transfusion. Sometimes it has been necessary to give as much as 2 to 3 liters of blood. We usually expose the internal carotid artery on the side of the lesion and occlude it temporarily by means of a rubber-covered Carrel vascular clamp, which is removed before the dura is closed.

The exposure offers no particular difficulties except when branches of the external carotid artery communicate freely with the aneurysm. From the arteriogram the location of the aneurysm and its feeding arteries can be determined with exactness. Needless to say, the flap should be so placed that all the contributing arteries are within reach.

When, as frequently happens, one or more branches of the anterior cerebral artery enter the aneurysm, the margin of the longitudinal sinus must be exposed, so that these branches can be clipped as they run on the mesial surface of the hemisphere.

Sometimes there are communications between dural vessels and the aneurysm, and the reflection of the dural flap must be made with

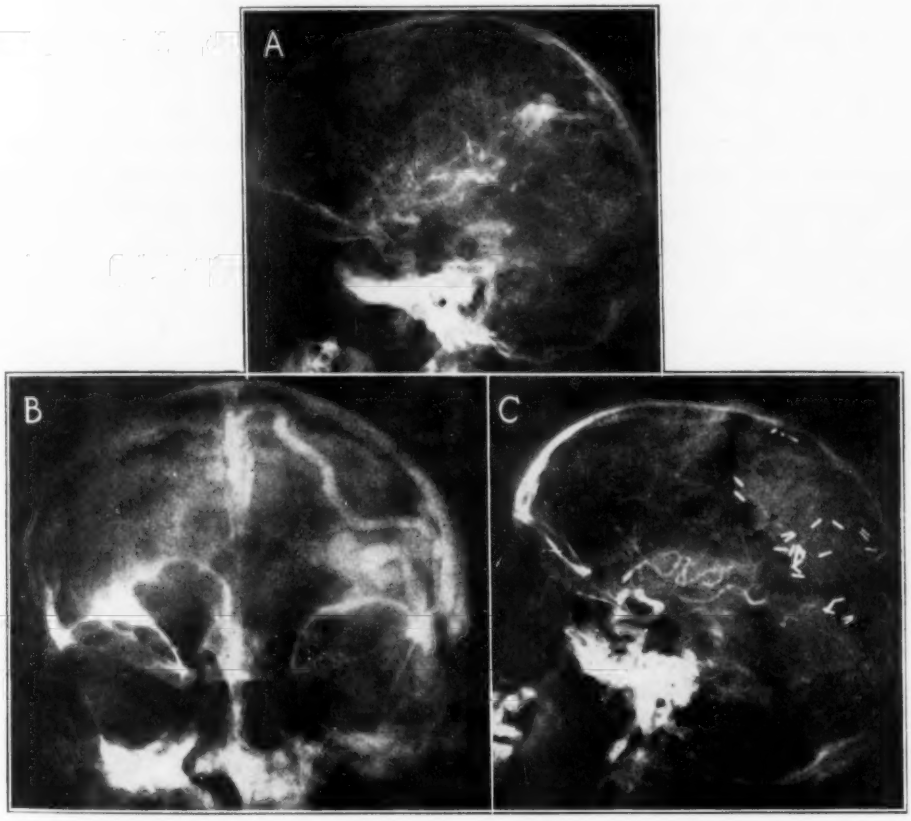

FIG. 13 (case 41).—Arteriograms in the case of a youth aged 19 with a history of two attacks of subarachnoid hemorrhage and moderate left hemiparesis during the past four years. *A*, arteriogram of the right internal carotid artery, showing arteriovenous aneurysm in the distribution of the right anterior and middle cerebral arteries. There is poor filling of the normal cerebral arteries. *B*, arteriogram of the right carotid artery two months after removal of the aneurysm. *C*, frontal exposure. Note the decreased size of the middle and, particularly, the anterior cerebral artery and better filling of the normal cerebral arteries.

circumspection to avoid tearing these vessels before they are clipped or coagulated. With the lesion exposed, the first thing is to locate the feeding arteries. Usually some of them are visible on the surface of the brain, where they should be clipped and divided. Sometimes they disappear beneath the cortex at some distance from the aneurysm, but

they can usually be distinguished from the normal cortical arteries by their size and the consistent hypertrophy of their walls. Next, the pia-arachnoid, as close as possible to the aneurysm, is divided at some point where the arteriogram indicates that the arteries coming from the deeper levels are most easily to be reached. It should be remembered that the aneurysm is wedge shaped and usually reaches almost to the ventricle, with numerous thin-walled arteries entering over its entire surface. The dissection is gradually carried deeper, being kept close to the aneurysm, and the entering blood vessels are caught with silver slips as they are encountered. Gradually, the incision of the pia-arachnoid is extended, care being taken to avoid the arterialized veins, usually two or three of which convey the blood to a dural sinus, in most cases the longitudinal sinus. When the apex of the pyramid has been sufficiently exposed, a blunt needle armed with a fairly coarse silk thread is passed around or through the stalk of the lesion. After the silk thread is tied, the ligated part of the stalk is cut, and more mass ligatures are applied as the apex of the wedge is dissected out. Hemorrhage is profuse until most of the arterial supply has been eliminated. As soon as the aneurysm has been dissected free to a sufficient extent to permit such a maneuver, it is grasped with a Brüning nasal clamp having a broad face. Thereby the hemorrhage from the aneurysm is greatly reduced, and with cautious pulling on the clamp more and more of the stalk is brought into view and caught with clips or mass ligatures. When the last arteries are obliterated, the aneurysm, formerly tense and pulsating, suddenly collapses, and the veins leaving the aneurysm become black. They can now be safely ligated and the lesion removed. Small arteries which have been torn in the process of mobilizing the aneurysm must then be sought and obliterated by clips or coagulation. They may be troublesome, as they are apt to retract into the white matter, but they must be found and eliminated at all costs. It is unsafe to rely on fibrin foam or similar means to take care of these small arteries; if possible, we try to catch them with silver clips. The clamp on the carotid artery is then removed, and after transfusion of a sufficient amount to raise the blood pressure to 100 mm. or a little more the cavity is again inspected for bleeding points.

Sometimes when the lesion is fortunately located, for instance, in the frontal or the occipital lobe, block resection of the lesion, together with a layer of normal brain tissue, can be carried out, thus greatly simplifying the procedure. Unfortunately, this is rarely possible, as most lesions are so close to the motor cortex that block resection is out of the question. Occasionally, when the malformation lies in the sylvian fissure, the dissection can be simplified by opening this fissure. The main branch of the middle cerebral artery will be observed to be greatly hypertrophied, and when the artery is followed from the anterior end of the sylvian

fissure toward the aneurysm, numerous small arteries going to the anuerysm may be seen and clipped as they leave the sylvian artery.

If the operation is performed shortly after an intracerebral hemorrhage, removal of the aneurysm may be considerably facilitated, for removal of the clot leaves a large cavity to work in. Moreover, part of the aneurysm is usually destroyed or thrombosed after the hemorrhage. It is important, therefore, not to postpone operation if hemorrhage has taken place. It should also be pointed out that in all cases of cerebral hemorrhage in which an arteriovenous aneurysm may reasonably be suspected arteriographic examination should be made, and if the suspicion is confirmed operation performed immediately.

SUMMARY

In a series of 3,202 verified cases of tumors of the brain there were 60 cases of arteriovenous aneurysm (1.8 per cent). Arteriovenous aneurysms are about twice as common in males as in females. In 5 cases the blood supply came largely through branches of the external carotid artery, although the aneurysmic malformation was actually located within the brain. In 2 cases the aneurysm was located in the distribution of the vertebral artery. In the remaining cases the blood supply came exclusively from branches of the internal carotid artery. In 1 case the combination described by Wyburn-Mason of an arteriovenous aneurysm in the retina and an arteriovenous aneurysm of the midbrain was observed.

The cardinal symptoms of arteriovenous aneurysm are epilepsy, subarachnoid hemorrhage and intracerebral hemorrhage with hemiplegia. A systolic murmur was observed in only 8 cases of the present series. It is always present when branches of the external carotid artery contribute to the blood supply of the malformation but is rarely noted when the aneurysm is located entirely within the distribution of the internal carotid artery. A typical calcification was observed in 6 cases.

The first symptoms usually occurred at a compartively early age, in the majority of cases during the second or third decade of life. In nearly half our cases the first symptom was an epileptic fit; in about a fifth, cerebral hemorrhage with hemiplegia, and in another fifth, subarachnoid hemorrhage. In the remaining cases uncommon or uncharacteristic symptoms were the first to be noted. Epileptic fits were not only the commonest initial symptom but the most constant symptom of all. Hemiplegia or hemiparesis, other than the transient hemiparesis sometimes observed immediately after an epileptic fit, occurred in about half our cases. Headache was a frequent complaint but was not usually characteristic. Attacks of typical migraine with scintillating scotoma were observed in 1 case.

Increased intracranial pressure was rare except with large aneurysms in the posterior fossa or immediately after hemorrhage. A choked disk was observed in only 25 per cent of cases. Mental changes were frequent, and in inveterate cases they were often severe. Epileptic fits, destruction of brain tissue by hemorrhage and short circuiting of the blood through the aneurysm are probable contributory factors in the mental derangement gradually taking place in many cases.

Encephalographic studies were carried out in most of our cases. The most frequent observation was cerebral atrophy, either general or localized. Sometimes the malformation was large enough to cause dislocation, suggestive of an expanding lesion. The dislocation in these cases was always slight and was sometimes combined with a variable degree of atrophy of the brain, a finding that must be regarded as strong evidence of an arteriovenous aneurysm.

The diagnosis of an arteriovenous aneurysm should always be confirmed by arteriographic study, preferably by the percutaneous injection of "per-abrodil." Both lateral and frontal exposures should be made.

With regard to treatment, we agree with Cushing and Bailey that ligature of accessible arteries visible on the surface of the brain is a useless procedure. Decompression is rarely indicated since pressure symptoms are habitually absent, and it may be contraindicated, as the presence of a cranial defect might increase the danger of leakage from the aneurysm Ligature of the carotid artery is a dangerous procedure; the risk of hemiplegia is great, and the effect on the lesion extremely uncertain. We have found no proof of the efficacy of roentgen treatment, and in 1 of our cases it could be shown that the lesion continued to grow in spite of roentgen treatment.

The choice lies between removing the lesion and leaving it alone. In case of the latter roentgen treatment may be tried, even if not much is to be expected from such therapy.

In our present series the aneurysm was removed in 24 cases, with 3 fatalities (13 per cent). With the addition of the 5 cases in our earlier series in which the lesion was removed, the mortality is reduced to 11 per cent. One third of the total number of patients operated on are practically well, with full, or nearly full, earning capacity; one third have defects, which in most had been present before operation, but sometimes were aggravated. These patients are partially able to work.

NOTE.—Between Sept. 1, 1946 and May 15, 1948, 14 arteriovenous aneurysms were removed, with 1 fatality, bringing the total number up to 43, with 4 fatalities, or a mortality of 9.3 per cent.

Serafimerlasarettet.

HEADACHE: A COMMON SYMPTOM OF CERVICAL DISK LESIONS

Report of Cases

AIDAN A. RANEY, M.D.

AND

RUPERT B. RANEY, M.D.

LOS ANGELES

THE SYMPTOM of headache constitutes one of the most perplexing diagnostic and therapeutic problems with which the physician is confronted.[1] It is also one of the most frequent complaints, accounting in one series, for example, for 24 per cent of more than 15,000 absences among factory employees.[1]

Several factors contribute to the frequency of the complaint, even when the condition responsible for the headache is not in the head.

The head is logically a region where functional complaints are centered. However, Friedman and others[2] wisely warned against diagnosing a headache as psychogenic purely on the patient's description of the pain, and they pointed out that emotional stress and inner conflicts readily exacerbate or precipitate not only psychogenic but migrainous and post-traumatic headaches as well.

Many anatomically uninformed persons consider the neck, except anteriorly, an imaginary plane separating the shoulders from the head. Therefore, if the ache or discomfort is above the shoulders, it is a "headache." Often patients complain of headache, and when asked to indicate

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Read at a meeting of the Los Angeles Society of Neurology and Psychiatry, Dec. 18, 1946.

From the Department of Neurological Surgery, University of Southern California, School of Medicine, Los Angeles.

1. (*a*) Friedman, A. P., and Brenner, C.: Post-Traumatic and Histamine Headache, Arch. Neurol. & Psychiat. **52**:126-130 (Aug.) 1944. (*b*) Horton, B. T., and Macy, D., Jr.: Treatment of Headache, M. Clin. North America **30**: 811-831 (July) 1946. (*c*) Friedman, A. P.; Brenner, C., and Merritt, H. H.: Management of Patients with Chronic Headache, J.A.M.A. **132**:498-502 (Nov. 2) 1946. (*d*) Grinker, R. R.: Neurology, ed. 3, Springfield, Ill., Charles C Thomas, Publisher, 1933, pp. 1065-1074.

2. (*a*) Friedman, A. P., and Brenner, C.: Psychomatic Treatment of Chronic Headache, Bull. Los Angeles Neurol. Soc. **11**:68-71 (March-June) 1946. (*b*) Friedman, Brenner and Merritt. 1c

its location, place the finger on the suboccipital or the upper cervical region. Perhaps, because "neck ache" is not euphonious and the term "pain in the neck" may convey an unintended meaning, "headache" is used in preference to these terms. If a close acquaintance is subject to headaches, the new sufferer may designate his discomfort by that term, when they are, more accurately, aches in the neck, jaw, face or other region.

Many persons subject to soreness, stiffness, aching or pain in the lower cervical, pectoral or brachial region decide that these symptoms are natural phenomena or that they are due to neuritis, rheumatism, arthritis or a cold settled in the neck. Being satisfied with the diagnosis and assuming the condition to be benign, the patient dispels such symptoms from his mind. If, however, the discomfort later extends to the head, the accepted diagnosis no longer suffices, and, fearing that the headache is a premonitory sign of serious illness, the patient complains to his physician of the headache and may never tell him of the important associated symptoms.

Another reason for the frequency of the complaint of headache is the fact that a deep-seated suboccipital pain may actually seem to the patient to be located in some part of the head, the cheek or the eye. If the patient is an intelligent and accurate witness to his symptoms, interrogation may disclose the site of the pain to be actually much closer to the suboccipital region than to the anatomic part in which he had believed the pain to reside. Discomfort in the vicinity of an old scar is often considered to be located exactly in that scar, and only detailed questioning may reveal that adjacent areas are as painful as the site of the old scar, ro even more so.

Many primary and secondary pathologic states are known to cause headache. Eight main types of headache, with more than one hundred subtypes, have recently been listed[1b] and many of these are the result of pathologic processes in regions other than the head.

PATHOLOGIC CONDITIONS IN THE NECK CAUSING HEADACHE

The importance of a pathologic process in the neck as a cause of headache is at times overlooked. Recognition that such a condition may produce headache is not a new departure in medical thinking. All agree that the suboccipital region and much of the scalp depend for sensation on the sensory roots of the upper cervical nerves, irritation of which may produce pain in the distribution of these nerves.

During the recent war, interest in the problem of headache stimulated one of us (A.A.R.) to make a special study of the frequent complaint of headache by the recently and slightly wounded soldiers. Investigation of the cases in which the recent injury could not be held responsible for

the headaches revealed that the patients were litter patients who, being interested in observing the activity about the hospital tent, held their heads up almost constantly for the first few hours and, consequently, strained the cervical muscles sufficiently so that the resultant discomfort caused them to complain of "headache." Heat applied to the muscles of the neck and the use of proper support for the head and neck afforded relief.

Regional splinting of a painful part by muscular contraction is a natural physiologic response. If the pain is persistent enough, the resultant muscular spasm may in itself be painful. Thus, a painful lesion in the cervical region may cause sufficient painful spasm of the occipital muscles to produce occipital headache. Hadden[3] and, evidently, Friedman and associates[1c] recognized the importance of relaxing the muscles of the scalp and neck in treatment of certain types of headache. Grinker[1d] mentioned indurative or rheumatic headaches as being occipital and suboccipital aches associated with tenderness of the neck and head. He also stated that occipitocervical myositis and cervical arthritis may cause symptoms referable to the head. Hadden[4] attributed certain types of headache and facial pain to fibrositis of the posterior muscles of the neck and scalp with involvement of the occipital nerves. Pollock[5] stated that diseases of the upper cervical vertebrae and pachymeningitis hypertrophica cervicalis may produce suboccipital headaches. Horton and Macy [1b] listed arthritis (including Pott's disease), hypertrophic pachymeningitis of the cervical portion of the spinal cord and tumors of the neck, the cervical segment of the vertebral column and the cervical portion of the spinal cord as causes of headache. Nielsen[6] listed cervical spondylitis as a cause of chronic headaches. Josey and Murphey[7] reported a case of surgically verified herniation of the sixth cervical disk in which the widespread distribution of pain included, in addition to a "pseudoangina" syndrome, pain located in the face, the back of the neck and the occipital region. In discussing the cervical rib syndrome, Eaton[8] mentioned that the patient may at times complain of pain in the face and head on the same side.

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3. Hadden, S. B., in discussion on Friedman and others.[1c]

4. Hadden, S. B.: Neurologic Headache and Facial Pain, Arch. Neurol. & Psychiat. **43**:405-408 (Feb.) 1940.

5. Pollock, L. J.: Head Pain: Differential Diagnoses and Treatment, M. Clin. North America **25**:3-13 (Jan.) 1941.

6. Nielsen, J. M.: Textbook of Clinical Neurology, ed. 1, New York, Paul B. Hoeber, Inc., 1941, p. 509.

7. Josey, A. I., and Murphey, F.: Ruptured Intervertebral Disk Simulating Angina Pectoris, J.A.M.A. **131**:581-587 (June 15) 1946.

8. Eaton, L. M.: Neurologic Causes of Pain in the Upper Extremities, S. Clin. North America **26**:810-833 (Aug.) 1946.

Thus, the differential diagnosis of the cause of the headache must include a consideration and investigation of the cervical region for evidences of these potential causes of headache. Actually, the differential diagnosis of some of these conditions is often difficult. Furthermore, the clinical picture may on one occasion be quite distinctive of one type of lesion and later become more characteristic of another type in the occipital or the cervical region. The most probable explanation of the overlapping of the clinical manifestations in cases of indurative and rheumatic headache, occipitocervical myositis, fibrositis and neuritis and cervical segmental hypertrophic arthritis is that in many cases seemingly falling into this categories there is one common underlying disturbance, namely a pathologic process of a cervical disk.

SPINOCAPITAL ACHE OR PAIN DUE TO PATHOLOGIC CERVICAL DISK

In this report we wish to single out and focus attention on the role of pathologic processes of cervical disks in the production of headache. When the lesion of the cervical disk is so advanced that the radicular pain and symptoms referable to the neck, because as these symptoms are patient usually fails to supply a complete history of headache, occipital pain and symptoms referable to the neck, because these symptoms are negligible in comparison with the pain in the arm or because he may have forgotten them or he considers such symptoms unrelated to his current ailment. When the pathologic condition of the cervical disk reaches the stage in which the pain in the extremity is severe, the symptoms in the head and neck often decrease, just as in cases of lesions of a lumbar disk the severity of the backache often diminishes when severe pain develops in the legs.

Headache is often caused by a lesion of the cervical disk, although the patient whose headache is due to such a pathologic condition often fails to volunteer information indicative of the true cause of his headaches. However, a thorough history in such cases usually reveals that the headache is closely associated with symptoms referable to the neck and that the underlying pathologic process is probably in the neck. Examination and roentgenograms disclose sufficient confirmatory evidence to establish the diagnosis.

History.—The patient complaining of headache must be asked to indicate with his finger the area affected. Even though he initially announces that his "head aches all over," after a few moments' reflection localization is usually possible. The "headache" may be located in the scalp, the face, or the cervical, suboccipital or other region. It is often unilateral but may be bilateral. The patient may state that it is located in the cheek, for example; yet detailed interrogation discloses that it "spreads" down across the jaw and ear to the base of the skull. It may

"spread" from the base of the skull up over the parietal to the temporal region. The radiation of the pain is an important, and particularly helpful, feature in diagnosis.

One must ascertain the anatomic location of any associated discomfort, pain, ache, soreness or stiffness, and determine whether or not it radiates from the face or scalp to the neck or suboccipital region, or vice versa. Other discomforts experienced prior to the onset of or during the course of the headache may clearly indicate a relation between the headache and the disturbance in the neck.

The patient must be questioned concerning various diagnostic terms under which he may have filed his discomfort. Otherwise, he may fail to remember important associated symptoms. One patient, for example, denied having numerous symptoms and diagnoses pertaining to discomfort in the neck but immediately admitted being subject to "colds settling in the neck" when the interrogator finally used that term, and the patient then admitted that the discomfort from this condition was often so severe that considerable medication was necessary for relief.

Headache due to pathologic changes in the cervical disks varies so greatly in intensity, frequency, location, radiation and projection that it cannot be clearly defined or classified. It may be constant, and variable only in intensity, or it may be periodic, occurring only on Tuesdays (when the housewife patient spends several hours with her neck acutely flexed, doing the ironing!). It may be mild and unnoticed except when exacerbated by nervous strain, muscular fatigue late in the day or a general irritable state and reduced pain threshold which may occur during the menses. It is important to know the frequency, duration and severity of the discomfort; the type of medication and the amount of the drug required for relief are a good index of the severity of the pain. It may be of short duration, occurring as a stinging, tingling sensation creeping up over the occipital or the parietal region of the scalp, or it may be thought of as a "sick headache," lasting twelve to seventy-two hours or longer. If severe, it may be associated with nausea or vomiting. Large doses of a medicament given for relief of the headache may be a precipitating cause of nausea, vomiting, visual disturbance or other symptoms. If severe, the pain may be only partially relieved by even large doses of the ordinary analgesics. Sedatives generally fail to give relief, although if the headache is treated with placebos or sedation, excessive fatigue may eventually cause the patient to fall asleep, and the resultant relaxation of the cervical muscles may make it possible for the sufferer to "sleep it off" and wake up several hours later without any discomfort. At times, however, sleeping with the neck in an unnatural or a cramped position may cause a defective disk to become symptomatic, and the patient awakens with "headache," "wryneck" or a "crick in the neck."

Physical and Neurologic Examination.—Examination of the patient whose headache is due to a lesion of a cervical disk reveals characteristic signs, if the examination is made at a time when the patient is having pain. If the pain is in the orbital or temporal region, the ipsilateral eye may be kept partially or completely closed. It may be noted that the motions in the cervical region are greatly limited; the patient may maintain his neck in a slightly tilted, rigidly fixed or "poker neck" position.

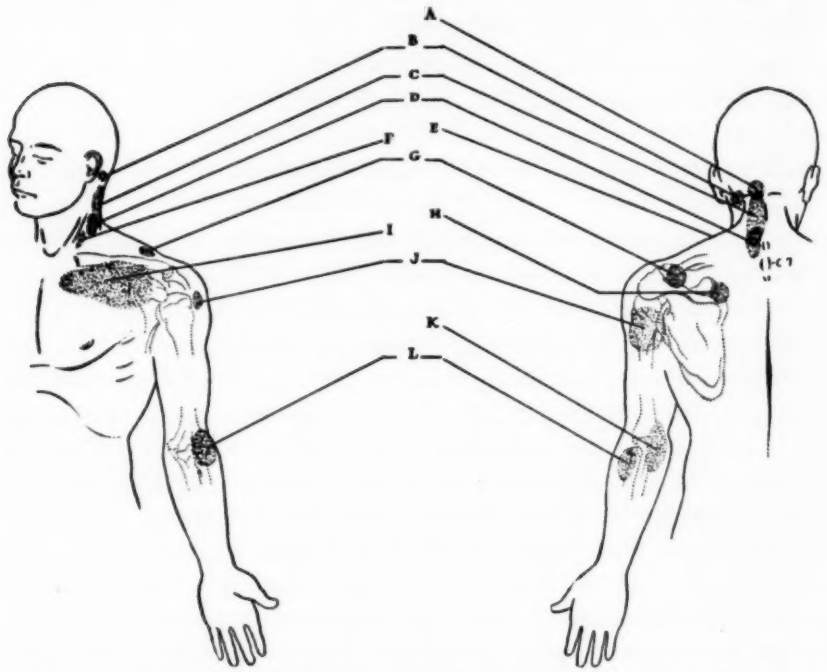

FIG. 1.—The shaded portions represent some of the more important areas of special tenderness found in patients having painful lesions of the cervical disks. *A* and *B* indicate suboccipital regions overlying occipital and auricular nerves and points of attachment of cervical muscles. C and *D* indicate paraspinal cervical muscles; pressure in this area may be transmitted to the nerve roots. *E* indicates tenderness of the muscles, and *F*, tenderness of the scalenus muscle. At *G*, irritated root fibers of lower cervical nerves are found to be acutely tender laterally if digital pressure is exerted in the scapuloclavicular angle; unless protected by thick muscles, the medial course also of these nerves is tender. *H* is the superior border of the medial end of the spinous process of the scapula; *I*, the superior portion of the pectoral muscles; *J*, the lateral deltoid region; *K*, the ulnar nerve, and *L*, branch of radial nerve lateral to the head of the radius.

The muscles of the neck may be spastic, and with cervical scoliosis or lordosis unilateral prominence of the cervical muscles may be present.

More important, however, are the points of tenderness in the suboccipital and cervical regions, the pectoral girdle and the upper extremities (fig. 1). Certain of these areas represent muscular soreness or tenderness. Others are in reality "lines of tenderness" indicative of the course of the irritated nerve root fibers in the brachial plexus or the peripheral

nerve. Discrete points of tenderness probably represent the location of the passage of the irritated fibers through the intervertebral foramen or hiatus of a fascial plane. In order to distinguish between normal sensitivity and abnormal tenderness, the patient may be asked to recall the tenderness of an inflammatory state or a sprain or to compare corresponding points on the opposite side of the body or, if the pain is bilateral, in other regions of the body.

When data from large numbers of cases are accumulated, it may be possible to determine the root affected by means of the physical examination alone, for the location of the tenderness depends on the site of the lesion. Irritation of the fifth cervical root, for example, cannot be expected to result in tenderness of the ulnar nerve, to which it ordinarily does not send fibers. The symptoms, as well as the clinical findings, vary also with the stage of development of the pathologic process. Minor lesions of the disk may not affect the nerve root sufficiently to produce radicular symptoms.

The neck compression test of Spurling and Scoville[9] is an important diagnostic procedure and is said to be almost pathognomonic. However, the patient's symptoms may be increased and prolonged if this test is not performed with care.

Another maneuver which we have found of diagnostic value is performed by applying head traction manually. Gently rolling the head slightly and assuring the patient that the procedure will not cause, but may relieve, discomfort aids in overcoming reflex or active contraction of the cervical muscles. If head traction is maintained for a minute or so, some relief from discomfort usually results in cases of a lesion of a cervical disk. It not only is an excellent diagnostic procedure in such cases, but also gives information as to the probability of relief by head traction.

The neurologic examination may disclose no detectable motor, sensory or reflex changes, just as early stages of lesions of lumbar disks may produce no such disturbances. Later stages of protrusion of the cervical disk, producing demonstrable sensory and reflex changes, are often associated with such severe pain in the neck, shoulder or arm that the patient is scarcely aware of headache. Because most of the case reports[10]

9. Spurling, R. G., and Scoville, W. B.: Lateral Rupture of the Cervical Intervertebral Discs, a Common Cause of Shoulder and Arm Pain, Surg., Gynec. & Obst. **78**:350-370 (April) 1944.

10. (*a*) Semmes, R. E., and Murphey, F.: The Syndrome of Unilateral Rupture of the Sixth Cervical Intervertebral Disk, with Compression of the Seventh Cervical Nerve Root: Report of Four Cases with Symptoms Simulating Coronary Disease, J.A.M.A. **121**:1209-1214 (April 10) 1943. (*b*) Bucy, P. C., and Chenault, H: Compression of Seventh Cervical Nerve Root by Herniation of Intervertebral Disk, ibid. **126**:26-27 (Sept. 2) 1944. (*c*) Eaton.[8] (*d*) Spurling and Scoville.[9] (*e*) Michelsen, J. J., and Mixter, W. J.: Pain and Disability of Shoulder and Arm Due to Herniation of the Nucleus Pulposes of Cervical Intervertebral Disks, New England J. Med. **231**:279-287 (Aug. 24) 1944.

are concerned with protrusions at a level lower than that of the fifth cervical vertebrae, the changes associated with protrusions of the lower cervical disks are fairly well defined, although, as suggested by Michelsen and Mixter[10e] the standard dermatome charts are in need of revision.

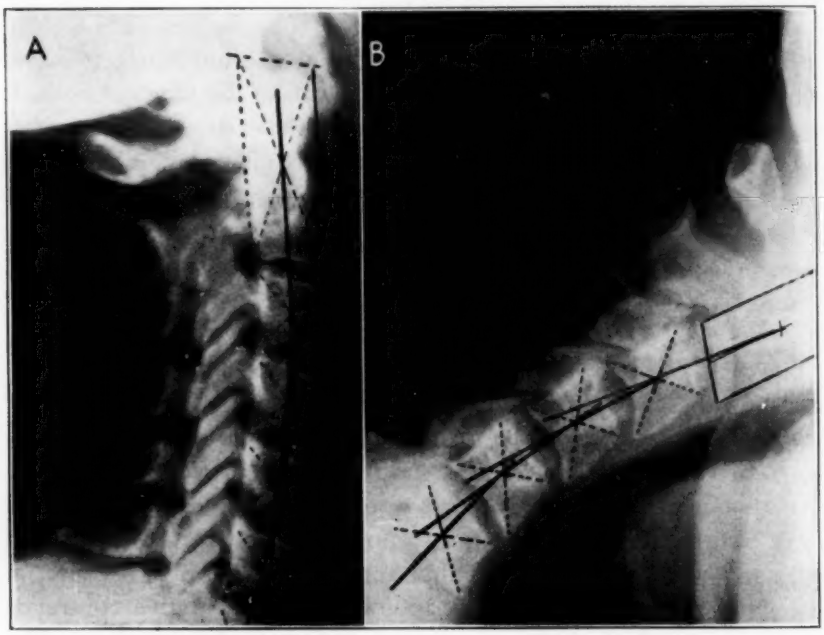

FIG. 2.—Roentgenograms of the normal cervical portion of the spine. *A*, lateral roentgenogram, showing slight lordotic curvature with the patient in a natural erect position.

By marking the film, as in this figure, optical illusions are eliminated and minor segmental alterations of curvature are readily identified. Normally, a "shingle appearance" results when straight lines connecting the centers of adjacent vertebral bodies are extended one segment caudally. The "center" of the space between the first and second cervical vertebrae is arbitrarily identified as the point of intersection of diagonal lines connecting opposite corners of a parallelogram the base of which corresponds to the inferior of the second cervical vertebrae and the anterior boundary of which constitutes a line extending from the anterior tubercle of the atlas to the anterior aspect of the epistropheus. Arthritic spurs, if present, must be disregarded in identifying the center of the vertebral bodies.

Hyperextension exaggerates the normal lordotic curvature.

B, in acute flexion of the cervical portion of the spine, a "shingle appearance" results when the lines are drawn, thus revealing a well distributed curvature.

The physical and neurologic findings in case of lesions of the upper cervical disks are even less well defined.[10a]

10a. Since this report was submitted for publication, revision of the dermatome charts has been accomplished by J. J. Keegan (Dermatome Hypalgesia and Posterolateral Herniation of Lower Intercostal Disc, J. Neurosurg. 4:115-139 [March] 1947).

Roentgenographic Examination. — The roentgenographic findings characteristic of lesions of the cervical disks have been well described by Whiteleather, Semmes and Murphey.[11] In the early stages of a pathologic process of the cervical disk lateral tilting of the cervical portion of the spine and segmental straightening or reversal of the cervical curve may be the only characteristic features. Narrowing of the disk space is of significance. Loss of the normal cervical curve is the most consistent abnormality.[12] The roentgenograms must be made at a time when the patient is having maximum pain if these abnormalities of alinement are to be demonstrated.

When the cervical portion of the spine is investigated roentgenographically for pathologic alterations in the disks, the initial view should be a lateral teleroentgenogram with the patient sitting in a natural position. The patient's shoulders may be depressed, but no manipulation of the neck should be permitted, because such manipulation may result in temporary relief or otherwise restore the cervical curve to normal.

All of the segments ordinarily participate in the cervical lordosis, kyphosis or straightening (fig. 2*A* and *B*), although there may be a gradual reduction in the degree of participation as the atlas or the first thoracic segment is approached. To find a normal roentgenogram of the cervical portion of the spine, one must first find a person whose history is completely negative for all symptoms of abnormality of this portion of the spine, and even then the roentgenogram may reveal asymptomatic congenital abnormalities and other defects. The "normal file" of the cervical region of the spine, as kept in some roentgenologic departments, often contains a relatively high percentage of abnormal roentgenograms of this region, and understandably so—the patient probably had symptoms referable to the neck (often of a lesion of the disk) or the films would not have been made.

Oppenheimer and Turner[13] showed that primary lesions of the intervertebral disk are a common cause of exostotic formations known as hypertrophic or deforming spondylitis. In cases of chronic lesion of the disk, bony proliferations, such as osteophytes, formation of spurs, arthritic lipping and "segmental hypertrophic arthritis," are betraying evidences of nature's attempts to heal the pathologic disks by physiologic immobilization. The presence of a narrowed disk with proliferation of bone must not be considered proof that that lesion is responsible for the symptoms. If the current symptoms are of only a few weeks' duration, it

11. Whiteleather, J. E.; Semmes, R. E., and Murphey, F.: The Roentgenographic Signs of Herniation of the Cervical Intervertebral Disk, Radiology **46**:213-219 (March) 1946.

12. Eaton.[8] Whiteleather, Semmes and Murphey.[11]

13. Oppenheimer, A., and Turner, E. L.: Discogenic Disease of the Cervical Spine with Segmental Neuritis, Am. J. Roentgenol. **37**:484-493 (April) 1937.

is quite possible that the disk at the level of the proliferation of bone is not responsible for the recently developed symptoms. Investigation of patients whose symptoms are of brief duration should be directed, rather, toward the study of disks at the level of which osseous proliferation does not exist because it is often at such a site that the cause of acute symptoms actually is found. The less dramatic improvement said to follow surgical treatment of a disk with arthritic changes[14] is possibly the result of a surgical attack on a defective disk, the chronic symptoms of which the patient had tolerated while nature was healing the defect by means of arthritic changes. Patients often ask for relief for chronic minor symptoms when severer acute discomforts demand medical attention.

Roentgenograms of the cervical portion of the spine should include views in the anteroposterior and the lateral plane as well as in the oblique plane. The oblique views are helpful, although none have been reproduced here because they lose much detail in reproduction.

It is obvious that arthritic changes in the anterior aspect of the vertebral bodies do not account for radicular irritation. Such changes do indicate a chronic pathologic condition of the disk, and the relationship of osseous proliferation and headache is so constant that when these roentgenographic changes are present it is rare indeed that a history of chronic symptoms referable to the head or neck is not obtained, unless there is adequate reason (hope of compensation from a recent injury, psychologic obscuration or a belief that "every one has stiffness in the neck and headaches") for such symptoms to be concealed.

REPORT OF CASES

Case 1.—H. H. A., a white man aged 47, had as his chief complaint headache in the left temple and the back of the head, which had been present since a recent injury to the head.

History.—Two weeks previously, as a result of a "Mickey Finn" and a blow to the left frontotemporal region, he had been unconscious for sixteen hours. On his regaining consciousness, he was observed to be bleeding from the left ear, and subsequently there was occasionally a watery discharge from that ear. For three days following the injury the headache improved gradually but then increased in severity. At the age of 22 he had been rendered unconscious for two hours by a blow to the right orbital area. He had had active pulmonary tuberculosis, but it had been arrested for twenty years.

Examination.—In the left frontotemporal area of the scalp there was a small abrasion with slight edema. The left ear drum membrane was hyperemic and perforated. The left eye was kept partially closed.

Points of special tenderness were noted in the temporal, suboccipital and paraspinal cervical areas on the left side. The lower left cervical roots and foramens were acutely tender. No sensory or reflex changes were detected.

Roentgenograms of the skull revealed no pathologic change. Roentgenograms of the cervical portion of the spine (fig. 3) revealed absence of the lower cervical

14. Whiteleather, Semmes and Murphey.[11] Josey and Murphey.[7]

curvature and narrowing of the disk space between the sixth and the seventh cervical vertebra, with pronounced hypertrophic lipping. Between the seventh cervical and the first thoracic vertebra the interspace was somewhat narrowed, and a small osteophyte was present posteriorly.

Course.—During the succeeding week his condition improved considerably with intermittent head traction and application of heat to the neck. There then developed mild otitis media and ear ache, distinctly differing from the previous discomforts and relieved after two days by local and systemic medication. During the following week, on three occasions for less than one-half hour each, he had

FIG. 3 (case 1).—Cervical lordosis, exaggerated in the upper segments, is absent at the lower levels. Note the narrow interspace and the large spurs at the level of the seventh cervical nerve root. A small osteophyte (arrow) and apparently a narrow interspace are seen at the level of the eighth cervical nerve root.

pain radiating from the shoulder to the third and fourth digits of the left hand. Slight hypesthesia of a dermatomal pattern affected these fingers. During the next few days he was asymtomatic and was discharged from the hospital.

Ten days later he requested rehospitalization for application of head traction because of headache in the left temporal area, pain in the neck and shoulder, radiating to the third and fourth digits of the left hand, and severe pain on the left side of the nose, the left nasomaxillary region and the upper second incisor tooth on the same side. He said that the pain in the side of the nose and the left upper

cervical region seemed to be produced by a steel spike projecting between the two areas. Within thirty minutes after application of head halter traction he was completely relieved of all pain except for slight soreness in the neck and shoulder. The pain in the left side of the nose, the cheek and the tooth could be reproduced in identical severity and distribution by moderate pressure with the finger at a point in the left suboccipital region about 2 cm. medial to the mastoid process.

During the succeeding week, traction was maintained fairly constantly but became less effective in affording relief, especially from the pain radiating to the fingers, and the patient requested surgical treatment.

Operation.—At operation (six weeks after the first examination) protrusions of the disks observed at the levels of the seventh and eighth nerve roots bilaterally were removed. At the level of the seventh cervical nerve root the protrusion was fairly firm except in the center. Both seventh and eighth cervical nerve roots and the left sixth cervical root, where a minor protrusion was encountered, were decompressed posteriorly by removal of bone.

Postoperative Course —After operation the patient had soreness in the infraclavicular area bilaterally and pain at the operative site. In addition, he complained of a cramp in the neck, which was due to spasm of the anterior scalene muscles. At times the pain in the scalene muscles became fairly severe and caused him to become frantic with fear that he would become unable to breathe. The discomfort often caused him to compain of a "choking sensation."

Convalescence was satisfactory until a violent sneeze precipitated an attack of severe pain in the upper left side of the chest and the left shoulder, associated with painful respiration. Investigation of the heart and lungs revealed no reason for the episode. The pain was concentrated in the left subscapular region and was most effectively alleviated by the application of heat. His convalescence was obviously retarded by this sudden trauma several days after operation. Two months after operation, he had at times some pain on the left side of the seventh cervical spinous process. Four months after operation he was asymptomatic except for occasional soreness in the left trapezius region.

COMMENT

Several cases of atypical facial neuralgia[15] due to lesions of cervical disks have been observed in which the pain could be reproduced by digital pressure in the suboccipital region, as in this case. The present case is a good example also of postconcussion or post-traumatic headache[15] resulting from injury to a cervical disk sustained at the time of head trauma. This case is one of the rare instances in which arthritic changes were present but the patient did not admit to a chronic history of symptoms referable to the disk. The patient's insurance claim has not been settled!

This case is of special interest because of the rather rapid progression of symptoms referable to a disk, from headache to severe radicular pain, necessitating surgical intervention.

Case 2.—Mrs. M. C. U., a white woman aged 31, had as her chief complaint, a severe headache, of eighteen months' duration.

15. A separate report dealing with cases in which symptoms of this nature were due to disk is being prepared for publication.

History.—The headache, located behind the right eye and in the right temple, often radiated across the side of the head to the right mastoid region and then down the neck. Its rather rapid onset was not preceded by injury. At times it completely subsided for a few days; at other times it became severe. When the headaches were so severe that she kept one eye closed, she had "double vision." Various drugs had been used, but none could be relied on for relief. At times she had dizziness, nausea or vomiting, but only when she had received medication for relief of the headache.

FIG. 4 (case 2.)—Reversal of the cervical curve with segmental angulation at the interspace between the fourth and the fifth cervical vertebra. Compare with the normal cervical portion of the spine (fig. 2*A*).

Three months previously there developed numbness and stiffness of the fingers of the right hand, but these symptoms subsided after two months. She often had pain and stiffness in the neck. At times she had a choking sensation in the right side of the neck (she placed her finger over the scalenus anticus muscle to identify the location).

During the examination, when the manual head traction test was being made, she volunteered the information that she had often thought that if only a device for supporting the head from the top of the car were available she would have been able to take automobile trips, which were impossible because of intense headache.

Examination.—The patient was mentally alert but appeared to be in distress. The head was slightly tilted, and she kept her right eye partially or completely closed. The "double vision" was found to be indistinct vision rather than diplopia. The right pupil was slightly irregular in outline. She permitted only guarded motion of the neck and head.

Points where only moderate degital pressure produced acute pain were on the right side in the suboccipital region, the paraspinal cervical muscles, the scalenus anticus muscle and the scapuloclavicular angle, and maximum tenderness was discovered over the foramens and roots of the fifth and sixth cervical nerves. There was also rather acute tenderness over the head of the right radius. Corresponding points on the left side were found to have no abnormal tenderness. Physical and neurologic examinations revealed otherwise an essentially normal condition. Studies of the spinal fluid revealed no abnormalities except in the protein content, which was 61 mg. per hundred cubic centimeters. The serologic reactions of the blood and spinal fluid were negative.

Head halter traction during the succeeding two days gave some relief.

Roentgenograms of the skull revealed an entirely normal picture. Electroencephalograms were reported to be suggestive of a deep-seated lesion in the right temporal area. Pneumoencephalographic studies revealed no evidence of an intracranial pathologic state.

Roentgenograms of the cervical portion of the spine revealed reversal of the cervical curve with anterior angulation at the interspace between the fourth and the fifth cervical vertebra (fig. 4).

Course.—Head halter traction maintained for six days after the pneumoencephalographic study afforded complete relief from all discomfort except for slight soreness of the neck. Being comfortable and relieved of fear that her symptoms were due to a cerebral tumor, she refused to have the traction. After being asymptomatic and ambulatory for two days, she was discharged from the hospital.

A week later, headache in the right temporal area and pain and stiffness in the neck developed. A molded Thomas collar was ordered, and during the week required for its manufacture she suffered so much from the pain in the head and neck that she remained in bed, using application of heat to the neck, with some benefit. As soon as she obtained the collar, she was able to be out of bed the entire day without discomfort. For a few weeks she used the collar fairly constantly when ambulatory. At the present time, almost five months since the collar was fitted, she uses it only on infrequent occasions when she experiences slight discomfort.

Comment.—Some of the features in this case suggested that the cause of the headache was intracranial. The patient, however, did not present the picture of mental dulness often associated with increased intracranial pressure. Not infrequently electroencephalographic tracings in cases of this nature are suggestive of a deep-seated lesion in the temporal or occipital region, at the site of pain. Perhaps such findings are the result of the influence on the tracing of involuntary contractions of muscle fibers in response to local pain. Pollock[5] stated that headache may be the presenting, but is rarely if ever the sole, symptom of intracranial tumor.

Case 3.—Mrs. F. G. I., a white woman, aged 35, complained of severe headache in the left temple and the base of the skull, of six hours' duration.

History.—The severe headache developed suddenly six hours previously, when she was writing at a low table while standing with her neck acutely flexed. The onset of severe pain was accompanied with faintness and blurred vision, which subsided when she walked several yards to a chair and sat down. She denied having had previous similar episodes or symptoms referable to the neck, and the only injury previously sustained was a minor one resulting from a traffic accident nine years before.

Examination —She was alert mentally and forcibly contracting the muscles of the face and neck, holding the head with the neck rigid. Head rotation to the left was limited to 10 degrees; other motions of the neck were less acutely limited.

FIG. 5 (case 3).—Loss of the lordotic curve, producing a "poker spine" appearance. Slight reversal of curvature is present at the interspace between the sixth and the seventh cervical vertebra. A small spur (arrow) is seen at the fourth cervical interspace.

Tenderness in the left temporal region was slight, but the left suboccipital area was more tender. Severe pain resulted from pressure over the lower cervical roots and foramens on the left side, as well as over the medial superior aspect of the spinous process of the left scapula. Any sudden movement of the neck caused severe pain in the temporal region, especially on the left. Although she complained of numbness of the hands, hypesthesia or hypalgesia could not be demonstrated.

Roentgenograms of the cervical portion of the spine (fig. 5) revealed a typical "poker spine." The lordotic curve was practically absent in the upper and lower segments, and there was slight anterior angulation at the interspaces between the fifth and sixth and the sixth and seventh cervical vertebrae. A small arthritic spur was present at the posterior inferior border of the fourth cervical vertebral body.

The spinal fluid was clear, and the pressure was 130 mm. of cerebrospinal fluid. The result of the Queckenstedt test, the cell count and the protein content were normal.

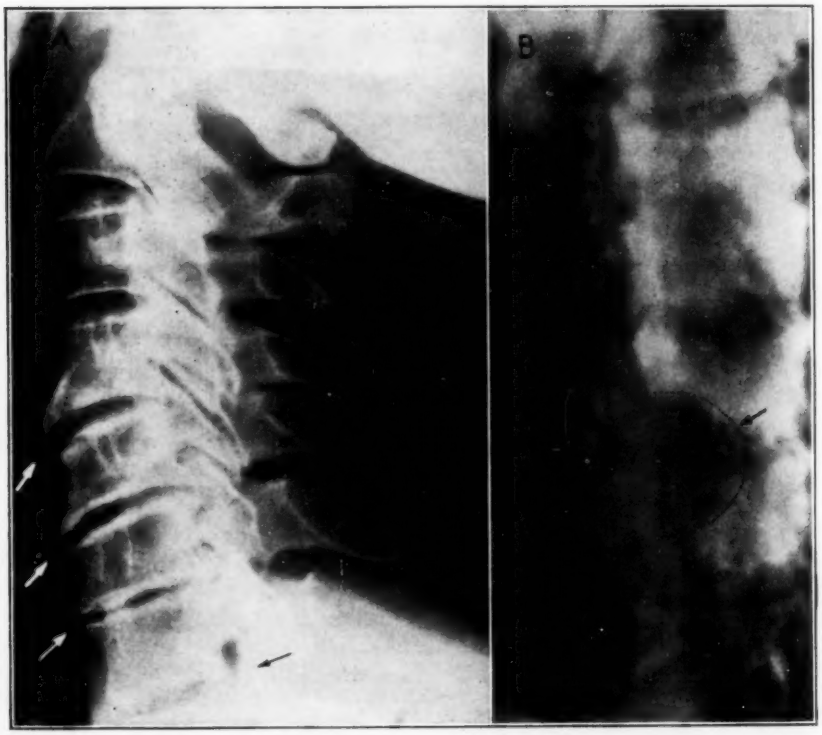

FIG. 6 (case 4)—*A*, pronounced narrowing of the disks, extensive arthritic changes and loss of cervical lordosis in the lower segments (white arrows). The space between the seventh cervical and the first thoracic vertebra (black arrow) shows little evidence of pathologic change; yet it was at this interspace that the large, acute protrusion of the disk was observed.

B, spinal studies with "pantopaque," showing a large filling defect (arrow) on the left side at the level of the eighth cervical nerve root. At the level of the seventh cervical nerve root on the right side, the angle of the superior root and the dura propria is obliterated. At other levels, slight ballooning of the root shadows can be seen, especially at the fifth and sixth cervical roots on the left side.

Head halter traction and heat to the cervical region were used, and after five days the patient was practically asymptomatic, although at that time there was tenderness of the left lower cervical nerve roots and the medial end of the spinous process of the left scapula. She still had some numbness of the hands; however, it could not be detected by sensory examination. Since then (ten months prior to the time of writing) there has been no recurrence of symptoms.

Comment.—Acute flexion and other extreme ranges of motion are relatively common precipitating causes of symptoms in patients who have defective disks. So-called postspinal headache[15] is often due to such a mechanism, rather than to the introduction of the needle into the spinal canal, the withdrawal of fluid or the effect of drugs used for anesthesia.

CASE 4.—Mr. O. P. O., a white man aged 67, complained chiefly of severe pain in the neck and the left shoulder, extending down to the left hand, of a few weeks' duration.

History.—For six years he had been subject to symptoms referable to the neck and pain in the shoulder, radiating to the left hand; however, the discomfort was never severe.

Examination.—The neck was held firmly fixed, and the head was slightly tilted to the right. There was tenderness on the left side in the lower cervical roots, in the scapular region and in the ulnar notch. Dermatomal hypalgesia, involving the two fingers in the ulnar distribution, was present.

Roentgenograms of the cervical portion of the spine (fig. 6*A*) revealed characteristic evidence of chronic pathologic changes in the disks at the fourth, fifth and sixth interspaces, but less evidence of a lesion of the disk between the seventh cervical and the first thoracic vertebra. On study with "pantopaque"[16] the most striking finding was a large filling defect on the left side of the level of the disk between the seventh cervical and the first thoracic vertebra (fig. 6 *B*).

Operation.—A large, soft herniation of the disk, which compressed the left eighth cervical root, confirming the roentgenographic findings, was removed.

Course.—During the nine months since operation, the patient has had no radicular pain, and the postoperative soreness and pain in the neck have subsided uneventfully. Hypesthesia of the fourth and fifth digits of the left hand still persists but causes him no concern. His only complaint is that of occasional pain in the left leg (ruptured lumbar disk?)

Additional History.—Because of the extensive arthritic changes indicative of chronic lesions of the disk and the frequency of headache as an early symptom of such lesions, it was suspected that the patient was, or had been, subject to chronic headache, although he did not volunteer such a history. He was not specifically questioned on this point until after operation, and at that time he admitted that for many years he had been subject to attacks of severe headache, which were usually preceded by numbness of the left hand. Within half an hour after the numbness began headache on the left side, and usually in the forehead, almost always developed. If he immediately went to bed, the headache would usually subside after a few hours.

Comment.—This man had tolerated for years the symptoms of the lesion responsible for the development of the extensive arthritic changes. The defective disks at these levels had never herniated as severely as did the disk removed surgically, and the spinal studies indicate that they protruded more laterally. The few weeks' duration of the acute symptoms of rupture of the disk between the seventh cervical and the first thoracic vertebra was insufficient time for development of extensive arthritic

16. "Pantopaque," manufactured by the Eastman Kodak Company, is a mixture of ethyl esters of isomeric iodophenylundecylic acids.

changes at that interspace. There is a history of headaches in almost every case of a chronic pathologic process of the disk, although it is usually not obtained unless specifically investigated, as in this case.

COMMENT

A defective cervical intervertebral disk may be asymptomatic. It has been shown that the interior of the disk is devoid of nerve fibers.[17]. If the annulus is affected, the fibers contained therein[18] may transmit painful stimuli, which, in turn, may be responsible for painful muscular splinting and spasm. This process, causing increased stress and strain on the articular surfaces, ligaments and tendons, may produce additional symptoms. Later stages of herniation of the disk, in which the nerve root is irritated or in which the spinal cord or its nutrient vessels are embarrassed, increase the symptomatic potentialities of an already complicated process.

Head pain due to pathologic cervical disk is not relieved by section of the root of the trigeminal nerve or by tractotomy or by both, even when the pain is confined within the zone made anesthetic or analgesic. Cervical sympathetic block usually does not affect the headache unless the procaine is poorly placed, so that it affects the cervical muscles and nerve roots, in which case some relief from the headaches may result.

Evidence presented by Davis and Pollock[19] indicates that the cervical portion of the sympathetic trunk does not contain afferent sensory pathways and that the sympathetic supply to the structures of the face and head consists purely of efferent fibers. In experimental work on cats they showed that pain resulted from faradic stimulation of the superior cervical sympathetic ganglion, even after section of either the posterior root of the trigeminal nerve or the posterior cervical roots. If, in addition to section of the posterior root of the trigeminal nerve, the posterior cervical spinal roots were sectioned bilaterally, pain responses to stimulation of the ganglion were abolished.

A satisfactory explanation of the mechanism by which a pathologic cervical disk produces headache does not necessarily include evidence of direct neural connection between the site of the pathologic disk and the region in which the patient believes the headache to be located. If, to the aforementioned complicated factors, one adds factors concerned in

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17. Jung, A., and Brunschwig, A.: Recherches histologiques sur l'innervation des articulations des corps vertébraux, Presse med. **40**:316-317 (Feb. 27) 1932.

18. Roofe, P. G.: Innervation of Annulus Fibrosis and Posterior Longitudinal Ligament: Fourth and Fifth Lumbar Level, Arch. Neurol. & Psychiat. **44**:100-103 (July) 1940.

19. Davis, L., and Pollock, L. J.: The Role of the Sympatheic Nervous System in the Production of Pain in the Head, Arch. Neurol. & Psychiat. **27**:282-293 (Feb.) 1932.

referred pain, diffusion of pain and mental projection of pain, then the mechanism by which a lesion in one region produces symptoms in another is more readily understood.

TREATMENT

The type of treatment required for headaches due to lesions of the disk depends on the severity of the symptoms. Many patients require only assurance that the headache is simply a symptom of a painful, but benign, condition which, in all probability, will never require more than conservative treatment. Patients suffering severe headache may require some type of immobilization of the cervical region, salicylates, nerve block or analgesics. Heat affords relief by reducing muscular spasm in many patients. Others may require head traction by means of a leather halter, or even a few weeks' skeletal-skull traction, followed by the use of a molded Thomas collar. Few patients have such severe headache and associated characteristic radicular signs that operation is necessary.

SUMMARY AND CONCLUSIONS

Many patients complain of headache when, in reality, the discomfort is in the neck. Not infrequently other symptoms are severer but cause the patient less concern than the headache offered as the primary symptom.

Headache has long been recognized as a symptom of a pathologic condition in the neck.

Lesion of the cervical disk is one of the commonest pathologic conditions of the cervical portion of the spine, and one of the commonest symptoms resulting therefrom is headache.

Headache due to pathologic cervical disk is generally associated with such symptoms that the responsible factor is suspected when a detailed history is obtained, and the diagnosis is established by characteristic physical and roentgenographic findings.

In adults, especially, lesions of the disk are sufficiently common so that not infrequently they are associated with other pathologic processes. Therefore, one must be cautious in assuming that the proved pathologic disk is the only lesion present unless it suffices to explain all the symptoms and signs.

Headaches following spinal puncture or head injury, for example, are often due to lesions of a cervical disk.

Reports of cases in which headache was due to a lesion of a cervical disk are presented.

1136 West Sixth Street.

SOCIOPSYCHOLOGIC STRUCTURE OF THE SS

Psychiatric Report of the Nurnberg Trials for War Crimes

LEO ALEXANDER, M.D.*
BOSTON

Associate Director of Research, Boston State Hospital; Instructor in Psychiatry, Tufts College Medical School

THE MOST important political organization in Nazi Germany was the SS (abbreviated from *Schutz-Staffel,* which means "protective quadron"). No totalitarian state can function without an SS-like organiation. It is therefore important for us as psychiatrists to know all we can about the SS, to understand its motivation and how it worked, what its strength was and what its weaknesses were; and it is the duty of sociologists, psychologists and psychiatrists to study these facts and to make them generally understood.

The SS was a criminal organization not only because its members actually committed crimes, but also because the essential mode of its thinking and its group behavior were those of all criminal organizations. If a member did anything which put his loyalty to the organization in a questionable light, either he was liquidated—killed—or he had to undertake a cirminal act which definitely and irrevocably tied him to the organization. According to the age-old custom of criminal gangs, this act had to include murder. In the SS this process of reenforcement of group cohesion was called *Blutkitt* (blood cement), a term which Hitler himself is said to have obtained from a book on Genghis Khan in which this technic of obtaining group cohesion was emphasized.

Examples of "blood cement" among prominent SS men were numerous. One striking example is that of Dr. Karl Gebhardt, a well known specialist in bone and joint surgery, professor of surgery at the University of Berlin and surgeon in chief of the renowned hospital at Hohenlychen.

This article is an abstract of a paper read at the seventy-fifth anniversary meeting of the Nederlandsche Vereeniging voor Psychiatrie en Neurologie, in Amsterdam, Netherlands, on June 12, 1947, and at the meeting of the Boston Society of Psychiatry and Neurology on Oct. 16, 1947. This paper will appear in full elsewhere.

* Formerly consultant to the Secretary of War of the United States, on duty with the Office of the Chief of Counsel for War Crimes in Nurnberg, United States Zone of Germany, 1946-1947; Lieutenant Colonel, ORC, Medical Corps, Army of the United States.

He held general's rank in the SS. He was suspected of having contributed to the death of Heydrich by failing to treat his wound infection with sulfonamide drugs. This omission made him politically suspect, and he was then expected to commit a crime which would dispel the suspicion against him and tie him more tightly into the SS criminal organization. The blood cement used in this case was a series of criminal experiments carried out on young girls captured from the Polish resistance movement, in whom he produced wounds complicated by tissue destruction and subsequently infected with gas gangrene bacilli. When a number of these victims died in spite of sulfonamide treatment, he thus proved that Heydrich's death was "fate determined" (a favorite SS phrase) and that he was not guilty of causing it. Gebhardt went one step further and involved the entire German medical profession in "SS blood cement" by presenting his report before a national medical meeting, where no objections were raised by the members.

Otto Ohlendorf, a lieutenant general in the *Waffen* SS and chief of the dreaded *Einsatzgruppe* D (one of the four special task forces which operated in the conquered eastern territories), admitted on the witness stand that he was responsible for the killing of 90,000 unarmed men, women and children between July 1941 and June 1942. Yet in 1939 this man had had a serious disagreement with Himmler on the latter's anti-Jewish policy, and in 1941 he had attempted to protect the persecuted anthroposophists. When he was asked why he had had to act thus against his own convictions by murdering innocent people, he replied that this was the general policy in dealing with men who, like himself, dared to disagree. The object was "to corrupt us morally."

Another general in the *Waffen* SS, Karl Wolff, described Ohlendorf as "an idealist, who, as *Reichsfuehrer* Himmler used to say, 'always ran about lamenting and deploring, and acting as if he were the only one who carried the Grail in his hands unsullied.'" Wolff continued: "Ohlendorf was rather soft by nature, to which the *Reichsfuehrer* objected and which he wanted to see changed by education. He had a blameless past, without dark points, so that he could not be put under pressure. The *Reichsfuehrer* wanted to harden this valuable man.

Viktor Hermann Brack, an official in Hitler's chancellery, after having been suspiciously anxious to release various people from concentration camps and insubordinately critical of the "winter catastrophe" in Russia, was entrusted by Himmler with the working out of plans for mass sterilization and mass extermination of Jews and conquered peoples.

Dr. Sigmund Rascher did not become the notorious vivisectionist of Dachau concentration camp and the willing tool of Himmler's research interests until after he had been forbidden to use the facilities of the Pathological Institute of the University of Munich because he was sus-

pected of having Communist sympathies. Then he was ready to go all out and to do anything merely to regain acceptance by the Nazi party and the SS.

Von dem Bach, as superior SS leader for East Prussia, in 1935 objected to the corrupt financial practices of the *Gauleiter* Koch, thus incurring the resentment of Goering and Himmler. He was arrested, and his execution was imminent, when Darré, a member of Hitler's inner circle, intervened and saved his life. He was not sent back to East Prussia but was given an equivalent assignment in Silesia. In 1940 he again incurred Himmler's displeasure by not cooperating in the establishment of ghetto camps for Jews and, later, by his failure to arrange pogroms in his territory. He was then sent to the Eastern Front and was put in charge of the so-called antipartisan forces. In this capacity he was responsible for mass executions of 366,211 Jews during 1942; and in August 1944 he was entrusted with the suppression of the Warsaw revolt, which resulted in the sacking and razing of Warsaw and the wanton murder of a large part of its civilian population. His orders were to take no prisoners. But von dem Bach incurred both Hitler's and Himmler's wrath by disregarding that order and taking General Bor-Komorowski and the remainder of his forces prisoners. He was ordered to report personally to Hitler's headquarters and explain his action. His conversation with Hitler was revealing:

> Hitler told me, "Actually you have forfeited your head because you have acted against my orders. But since your method has been successful and the battle is won after all, I have accepted it." That was typically Hitler. But I still felt uneasy, because I could name innumerable persons to whom he gave a decoration and then had them hanged. He continued: "The success was decisive: I have approved of it now. You were lucky. Maybe you will be lucky again the next time, too. You seem to have a streak of luck now." Everybody was there—Keitel, Himmler, Goering, all prominent people. I said, "I have held out and been successful only because I kept up negotiations with the partisans." Hitler then said, "Hungary threatens to secede. I shall send you now to Hungary to prevent the secession of Hungary, and I shall give you plenipotentiary power. You have to prevent Horthy from jumping off the running train—if need be, by armed force. The moment of action I shall determine myself. I shall send you word by courier. You will have to arrest the entire Hungarian government and to take care that the Hungarians do not fight against Germany as the Rumanians did."

In the course of the *coup de'etat* in Hungary in October 1944, the regent of Hungary was imprisoned, his son was kidnapped, 60,000 Jews were arrested to be deported or exterminated, of whom 25,000 were killed outright or perished on the subsequent eight day death march.

Von dem Bach revealed real insight into the sociopsychologic role that involvement in crime played in Nazi Germany when he said:

> There were opposition groups in Germany. Compelled by the war and by their bad conscience, they were brought together. Hitler let them all become guilty. I am firmly convinced that his great crimes had only that one motive—

to let them all become guilty. They must never be allowed to jump off the running train. If you see it that way, you see how one becomes guilty.

In the lower echelons of the SS, the principal places within the confines of Germany where members could acquire blood cement were the concentration camps. When after this conditioning experience SS members were considered reliable enough, they were sent abroad into the occupied countries, where they were called on to perform similar crimes inside and outside specific camp areas.

There were instances in which men ended by committing the very crimes they had been most vocal in disapproving: Dr. Gerhard A. H. Rose, vice president of the Robert Koch Institute, in Berlin, in regard to the typhus experiments on prisoners, and Col. Karl von Bothmer, in the case of the shooting of civilians without trial in occupied countries, are significant examples. In such instances the acts were obviously the result of group pressure, although no overt coercion was brought to bear. These cases illustrate that an important fact concerning motivation with which one is familiar in ordinary crimes applies also to war crimes and to these ideologically conditioned crimes against humanity; namely, that fear and cowardice, especially fear of ostracism by the group, are often more important motives than simple ferocity or aggressiveness.

The master crime to which the SS was committed was the genocide of non-German peoples and the elimination by killing, in groups or singly, of Germans who were considered useless or disloyal. In effecting the two parts of this program, Himmler demanded and received the cooperation of physicians and of German medical science. For this trend of research in Nazi Germany, namely, that toward development of scientfic methods of destroying and preventing life, I have proposed the term "ktenology" the science of killing.

In the course of this ktenologic research, methods of mass killing and mass sterilization were investigated and developed, as well as methods for rapid and inconspicuous individual execution. A committee of physicians and medical experts, headed by Dr. Karl Brandt, developed various methods of extermination by gas. At first carbon monoxide was used; later, cyanide gas ("cyclon B"), with occasional use of warfare gases, for which this program supplied human experimental material. Of the individual methods of inconspicuous execution, which were usually carried out in camp hospitals by medical personnel, the most widely used was the intravenous injection of phenol or gasoline. This, however, left a tell-tale odor with the corpse, which made it an undesirable means of executing prominent prisoners or high-ranking Nazi party personnel, for which secrecy was essential.

The triumph of that part of ktenologic research aimed at finding a method of inconspicuous execution which would produce autopsy findings

indicative of death from natural causes was the development of intravenous injections of a suspension of live tubercle bacilli, which brought on acute miliary tuberculosis within a few weeks. This method was produced by Prof. Dr. Heissmeyer, who was one of Dr. Gebhardt's associates at the SS hospital of Hohenlychen.

As a means of further camouflage, so that the SS at large would not suspect the purpose of these experiments, the preliminary tests for the efficacy of this method were performed exclusively on children imprisoned in the Natzweiler concentration camp.

It is clear from the foregoing instances that one is dealing here with a generalized eruption of aggressive, destructive activity which invaded all spheres of life, including the physician's office. The resulting attitudes brought about a peculiar perversion of the concept of death, a perversion which can be described as an idolatrous delight in death, for which I have coined the word "thanatolatry."

This heathen concept, somewhat reminiscent of voodoo beliefs, expressed itself in many ways. The blood and soil theory is the best known example. According to this theory, formulated by Rosenberg and Darré, and widely accepted in Nazi Germany, soil in which people of Germanic race were buried itself became German and, in turn, also could give German characteristics to people who nourished themselves from that soil. Goering's statement, made early in the war, that "with every German airman who is killed by the enemy our *Luftwaffe* becomes stronger" is another example.

Dr. Karl Brandt, plenipotentiary in charge of all medical activities in the Reich, when asked about his attitude toward the killing of human beings in the course of medical experiments, replied, "Do you think that one can obtain any worth while fundamental results without a definite toll of lives? The same goes for technologic development. You cannot built a great bridge, a gigantic building—you cannot establish a speed record without deaths!"

In a similar vein, many SS men took a curious pride in the fact that even in peacetime they had many fatalities during "realistic" military training. Human bodies were encased in the concrete of fortifications and bunkers, as though such bodies could give strength to inanimate matter. Himmler himself stated the belief that strength could be derived from inflicting deaths and remaining unmoved by it.

It is one of the laws of psychology, which is in harmony with similar, more general, physiologic principles, that destructive urges of great magnitude and depth and destructive concepts arising therefrom cannot remain limited or focused, but must inevitably spread and be directed against one's own group, and, ultimately, against the self.

Examples of this self-punitive consequence are the murderous and hateful relationships which finally broke out among the ranks of the SS themselves. This ultimately and inexorably brought down the destruction of the group and of the individuals who composed it—a destruction expressed in the marked turnover of personnel, many of whom found their deaths in SS bunkers at the hands of their former comrades. They were those who lost in the constant fratricidal struggle, which was euphemistically termed "the self selection of leaders" (*Fuehrer-Selbstauslese*).

There is an "all or none" law of fundamental psychologic attitudes. A man cannot for long be criminal in one type of relationships and decent in others. The destructive drive, once unleashed, is bound to engulf the whole personality and occupy all its relationships. Himmler himself, with a remarkable degree of insight, summed up the whole case when he said in a speech, on Oct. 4, 1943, to his generals, "We have become—I say that now behind locked doors, and this is intended only for this small circle—a very corrupt nation."

We must see in the destruction of the morals, the institutions, the ideals and the cities of Germany the ultimate realization of this destructive drive which was in no other organization of the Nazi state so limitlessly unleashed as it was in the SS.

The question now arises whether these destructive drives form the main motivation of Nazism or whether they were merely superimposed on, or formed the superchargers of, other aims and goals. There was, of course, the one over-all conscious aim and goal—to gain power and complete control, first over the German people, then over Europe, and finally over the world, which was in itself a basically destructive and aggressive conception.

The determining elements of Nazism, apart from the fundamental aim and goal of complete domination for Germany and genocide of all non-Germanic peoples, were not any additional specific articles of doctrine or contents of thought, but, rather, certain specific attitudes or modes of thinking. The most important and basic element of these attitudes was an intense hostility against spiritual and rational modes of thinking and behavior.

This Nazi hostility toward spiritual and rational attitudes is the same basic hostility to spirit, culture, intellect and reason which is inherent in the unconscious mind where this and other destructive drives are dormant. All these destructive drives are repressed and sublimated in the mature individual as long as he acknowledges guidance by spiritual and rational values. The disruption of spiritual values and the value of reason is, therefore, one of the prerequisites for release of primordial destructive drives in the adult.

It is not surprising, therefore, that the Nazis, in their effort to release and utilize these drives, made a concerted attack against these two inhibiting forces, spirituality and reason, which in the mature individual and in mature civilized societies prevent the eruption of the primeval destructive drives.

The first open attack was against the religious-spiritual and humane ideals of the superego, which were derided as weaklings' and slaves' morality; and a new "strong man's religion," the SS religion with heathen trappings and Hegel's worship of the strong, was substituted.

A systematic campaign of propaganda to break down superego values was directed at the entire population, including children. A ghastly example is a widely used school book on applied mathematics, on the high school level, entitled "Mathematics in the Service of National-Political Education.[1] The choice of problems in this book on mathematics was designed to break down the taboo against mass killings by formulating the problems in terms of mass killing, as when (page 75) pupils were asked to compute how much phosgene would be needed to poison a city of a certain area and of a specified population, with special attention to the average rate of breathing in relation to the atmospheric concentration of the gas. The book also attacked normal and desirable compassion for the sick and crippled by setting problems in terms of distorted statements of the cost of their proper care and rehabilitation, asking, for instance, how many new settlement houses could be built and how many marriage allowance loans could be given to newly wedded couples for the amount of money it cost the state to care for "the crippled, the criminal and the insane."

The taboo against promiscuity and disruption of family life was broken by the notion of the "fountain of life" (*Lebensborn*), which was to flow freely in the interest of the "state." Thus, the religious-humane-cultural superego common to all civilized peoples was replaced by an exclusively tribal superego. The psychiatrist stands in amazement before the thoroughness and completeness with which this perversion of essential superego values was accomplished in adults.

The explanation of this phenomenon is twofold. First, it may be that the decisive importance of childhood and youth in the formation of essential superego values may have been overrated by psychiatrists in a society in which allegiance to these values in normal adult life was taken too much for granted because of the stability, religiousness, legality and security of the nineteenth and early twentieth century society. It may be

1. Dorner, A.: Mathematik in Dienste der Nationalpolitischen Erziehung: Ein Handbuch für Lehrer, herausgegeben in Auftrage des Reichsverbandes Deutscher mathematischer Gesellschaften und Vereine, ed. 2, revised, Frankfurt a. M., Moritz Diesterweg, 1935, pp. 1-118; ed. 3, revised, 1936, pp. 1-118.

that the lesson learned in youth has to be continually relearned in adult life in order to be durable, and that the ideals acquired in youth must be continually striven for again in order to remain valid and desirable.

On the other hand, however, it must be admitted that probably much more of the old tribal superego has remained alive in Germany than was noticeable on the surface. It was never thoroughly replaced by the religious and humane-cultural superego which other civilizations accepted more wholeheartedly. Therefore, the shift of superego values toward the exclusively tribal ones was easier in Germany than it would have been in other nations. Be that as it may, it is definite that this regression of superego ideals from the religious-humane to the tribal constituted the most important step in the nazification of Germany; and it is obvious that the opposite step would be the most important one in the denazification of Germany.

The next, more insidious, attack was against all modes of rational thinking and behavior, and, indeed, against reason itself. Reason and logic were decried as cold intellectualism; reasonableness was regarded as weakness. Teachers and educators conspired in the general campaign against logical thinking. Nazi school books are full of *non sequiturs,* as if to train children in illogical thinking.

With moral, or superego, values perverted and reason discredited, the stage was set for the direct and uncontrolled release and the conscious encouragement of the unleashing of instinctual drives. This encouragement was given in the form of seduction. The more subtle form of this seduction was a peculiar cult glorifying the pleasurable satisfactions derived from the release of instinctual energy which was established.

Conscious destructive thinking in regard to all interhuman problems gained more and more prevalence among all those who became "infected" with Nazi ideology. The continuation of thinking in terms of destruction as the solution of all problems is today the best way to tell a Nazi from a non-Nazi.

All this disastrous propagation of destructive thinking was a means of preparatory seduction to destructive activity. This was early combined with more direct seduction to destructive physical activity calculated to let the novices taste the primitive satisfactions obtained from regression to infantile-sadistic patterns and to keep that taste alive in the initiated. It was for this reason that the training and indoctrination centers of the SS were always attached to concentration camps.

People can be more easily seduced to lower levels of emotional expression than educated to higher ones. Group and mass sanction played contributing roles in the process of seduction. Especially during initiation, the Nazis always saw to it that the worst atrocities, the so-called *Sonderaktionen,* or special actions, were always committed by groups.

The individuals who, once they were in the SS or similar formations, did not succumb to this "group polarization" were few.

However, the over-all result that Germans thoroughly infected with Nazism not only could, as Himmler himself expressed it, derive strength and joy from the act of killing but could gaze on heaps of 100, 500 or a 1,000 freshly killed corpses with frank enjoyment, or with a veneer of rationalization, was the combined effect of indoctrination, seduction and sanction, or, expressed in pavlovian terms, conditioning.

One of the main sources of sanction was the group, in particular, the participating group. However, a sinister type of sanction came from a surprisingly large circle of ostensibly nonparticipating women, especially women close to SS and other activist circles, although not themselves enrolled in the SS or other activist organizations. Dr. von Baeyer[2] has pointed out this role of women in Nazi society with great clarity.

In summary, we found that there was a widespread release of destructive drives and destructive activity in the German people under the Nazi regime caused by the combined effects of: (1) indoctrination with antispirituality, resulting in a shift of superego values away from the religious-spiritual-humane ideals toward the exclusively tribal (the *Deutschland über Alles* doctrine); (2) indoctrination with antirationalism, resulting in the discrediting and abandonment of reason as the main instrument for decision by individuals; (3) seduction, resulting in pleasurable release of repressed instinctual drives; (4) sanction—namely, group sanction and sanction by the nonparticipating larger part of the female population—resulting in a pleasurable absence of guilt feelings. Observation of the Germans during the Nazi regime indicated that they represented a group in which instinctual destructive drives can be more easily released than they can be in most population groups under comparable conditions of indoctrination and seduction by propaganda. I wish to emphasize at this point that I consider this trait not at all as a biologic or racial characteristic, but as the result of social, cultural and political conditioning. I am, therefore, using the word "Germans" or "German people" in this connection as a term denoting members of a specific social, cultural, national, traditional unit—the German state or community—and not as a term denoting descendants of a particular racial or familial ancestral strain.

The conditioned aggressive behavior of the Germans was not a natural overflow of excessive ferocity but had to do with a more complicated process concerned with the higher levels of integration—the psychic control mechanisms rather than the instinctual drives themselves. It is, indeed, in these control mechanisms rather than in the instinctual drives

2. von Bāyer, W.: The Emotional Psychology of the German Woman Under the Nazis, unpublished manuscript.

themselves that one has to look for the reason for this diastrous German trait. When one examines these psychic control mechanisms one finds the greater degree of difference between members of the German cultural sphere and those of other, especially Western, cultural groups.

First of all, the social-philosophic *Weltanschauung*—in other words, superego notions—has a greater guiding value to the German than to members of other cultural groups. This trait, of course, has its asset side. If the superego values believed in are good—for instance, religious, spiritual and humane—then the particular German has a greater chance of becoming a saint than have members of other groups. The only trouble with predominantly superego-determined guidance of the personality is that the superego, even at best, is the least deeply anchored part of the personality, and therefore the least stable and reliable unless it is continually reenforced by external social-moral forces, such as religion, law and public opinion. The superego structure is, therefore, in peril whenever these established guiding forces weaken or are in the process of being undermined, shifted or perverted, and becomes itself open to undermining, shifting or perversion even in adult life—a fact which is probably more important than we have been aware of heretofore.

The most important deficiencies of the members of the German social-cultural system, however, lie in that part of the personality the function of which is to deal rationally with reality in a responsible and relevant manner—that is, in the ego. A great deal of work has been done with reference to the ego structure of persons bred and integrated into the German social-cultural system. Brickner,[3] especially, pointed out clearly that one of the outstanding features of the German culture is a resentment of reality and a denial of it, which he compared to the attitude of the paranoid person in the reference to reality. A propensity for fanatical, and even delusional, thinking in Germans is, indeed, striking to those who come in contact with them. It exists not only among the defendants in the recent trials but also in the population at large.

Linked with this denial of reality is a widespread antirationalism, obviously originating as a spontaneous defense, but worked up into a policy designed to crush any attempt at exercise of the basic ego functions which might interfere with the denial of reality. With this unrealistic and antirational attitude go an unwillingness and an inability to accept personal responsibility.

A fourth trait is probably a consequence of the other three. This is a disinterest in the relevant aspects of life, with a preference for certain irrelevant ones.

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3. Brickner, R. M.: Is Germany Incurable? Philadelphia, J. B. Lippincott Company, 1943.

In other words, one has here the result of a social and cultural molding of the German personality in which, somehow, the ego part of the personality has been insufficiently developed or has been crushed by educational, social or other forces. What is left is a weak ego with a vastly overdeveloped but, of necessity, unstable superego, which is superimposed on this weak ego and on a probably normal id portion of the personality. The emptiness of the ego sphere is the most striking finding which differentiates members of the German cultural group from members of other cultural groups. With this weakness of the ego, of course, goes the need to receive the main motivated stimuli from spheres of the personality other than the ego—that is, from both the superego and the id.

One of the most sinister traits of the Hitler regime was that it utilized a craving for irrelevant excitement by these personalities with weak ego in its system of seduction already described. It was, apparently, a function of the more spectacular atrocities in concentration camps, especially in Auschwitz, to provide this type of excitement. The most spectacular of the mass atrocities were called *Sonderaktionen* ("special actions"). One of these, practiced particularly in Auschwitz, was the burning of live prisoners, who were often children, in pits measuring 20 by 40 by 50 meters, on piles of gasoline-soaked wood.

The reaction of the SS to the ghastly shows offered and eagerly watched at Auschwitz was a peculiar mixture of enjoyment and detachment. Most revealing of this reaction is the diary of Prof. Dr. Hanns Hermann Kremer, a most interesting source of psychiatric documentation.

In 1941 Professor Kremer went on active duty as medical officer in the *Waffen* SS. His diary includes entries made during his tour of duty in Auschwitz from Aug. 30, to Nov. 18, 1942. It is a ghastly document, void of anything but the most primitive instinctual expressions, consisting of accounts of primitive sadistic enjoyment of mass astrocities, of the simple oral gratifications of eating (with enumerations of all the dishes consumed), of accounts of his own pseudoscientific atrocities (consisting of the removal of vital organs from living prisoners) with its satisfactions of the curiosity reflex achieved without strings tied to it and in primitive infantile fashion, and last, but not least, of the greedy primitive satisfactions of collecting and shipping all kinds of valuable items—with the appraisal value recorded for each parcel—to his home in faraway Münster. The spectacular mass atrocities, of which he attended fourteen, the numerous simple executions, inflictions of punishment, his own pseudoscientific atrocities and the enjoyment of food and wine are all related in the same vein, showing a peculiar mixture of detachment and of the eager excitement of a child attending a party.

The following entries are quoted:

September 2, 1942: First time present at a *Sonderaktion* [special action] at 3 in the morning. Compared with this, the *Inferno* of Dante seems to me a comedy. Auschwitz is not called for nothing the "camp of extermination."

September 5: This afternoon present at a *Sonderaktion* from the female concentration camp. The most horrible of horrors. *Hschf.* Thilo, doctor of the troops, is right when he told me this morning that we are at "anus mundi." In the evening, at approximately 8 hours, again present at a *Sonderaktion* from the Netherlands. Men all want to take part in these actions because of the special rations they get then, consisting of a fifth part of a liter of *Schnapps*, five cigarets, 100 Gm. of sausage and bread. Today and tomorrow on duty.

October 16: Today I sent the second parcel, 300 reich marks worth, to Mrs. Wizemann to save it for me (soap, soapflakes, prepared foodstuffs). Had taken a photograph of a syndactylous Jew in the camp (father and uncle have the same disease).

October 17: Present at an infliction of punishment and eleven executions. Took out living, fresh material of liver, spleen and pancreas after injection of pilocarpine.

October 18: In wet cold weather present at the eleventh *Sonderaktion* (Netherlanders) on Sunday morning. Shocking scenes with 3 women, who beseech us for the naked life.

November 8: Took part in two *Sonderaktionen* last night in rainy weather (twelfth and thirteenth). In the afternoon, another *Sonderaktion*, which was the fourteenth I have taken part in. In the evening we had a nice time at the leaders' club, invited by *Hstuf.* Wirths. We had Bulgarian red wine and Croatian plum *Schnapps.*

I am of the opinion that the fundamental disturbance in the social-psychologic structure in Germany is an ego disturbance. Where does it come from? We know, especially from our experience in military psychiatry, that anxiety is one of the most important ego-disrupting forces, and we have learned about its relation to aggressiveness: Aggressiveness generates anxiety, and anxiety, in turn, generates aggressiveness. There is suggestive evidence that anxiety is, indeed, a more commonly predominant feeling in Germany than elsewhere, in children as well as in adults. Investigations are necessary to determine whether this anxiety relates to historical circumstances, to geographic circumstances, or whether it is fundamentally based on the structure of the German family.

What are the practical measures which can be taken in reforming the social psychologic situation in Germany? First of all, further studies are needed. I hope that in these considerations I have indicated a few leads to further research. Yet already we have learned enough to know that, most of all, mature guidance on the part of the free nations is needed. For that purpose, we of these nations must maintain our own maturity. Maturity is a level hard to maintain. Regression is all too easy and pleasurable, and the danger is that our German patient may again forsake

development of his ego in order to submit once more to another ephemeral neo-superego, with a new orgy of release of instinctive destructiveness.

In spite of the magnitude and extent of the problem, we should not allow ourselves to become discouraged. All constructive, forward-looking forces are needed in the task, and we, as psychiatrists, will have to do our share. I think that we psychiatrists have made a fairly hopeful start as potential contributors to social health. Lifting our eyes from the individual mental patient in front of us, we have proceeded to contribute helpful understanding to family relationships, especially the child-parent relationship, then to the teacher-child relationship, then to the schools in general, to the courts and, most recently, to armies at war in terms of morale. It is now time that we make every effort to contribute helpful understanding to national and international politics.

METASTATIC TUMORS OF THE CENTRAL NERVOUS SYSTEM

I. Intracerebral Metastases as the Only Evidence of Dissemination of Visceral Cancer

CHARLES RUPP, M.D.
PHILADELPHIA

METASTATIC Tumors of the central nervous system are not uncommon; their diagnosis is often difficult, and the pertinent literature, as Globus[1] commented, is relatively meager. It therefore was thought worthy of interest to review the material in the laboratory of neuropathology of the Philadelphia General Hospital. These cases are drawn not only from the neurologic and neurosurgical wards but from the medical, surgical and radiologic services as well.

The cases of metastatic involvement of the central nervous system have been grouped on the following basis.

GROUP 1.—Forty-two cases in which the metastatic involvement apart from extension to the regional lymph nodes, was observed only in the brain in the form of discrete metastatic nodules, either single or multiple, ranging in size from 1 to 9 cm. In this group the clinical signs and symptoms simulated those of a primary intracerebral lesion.

GROUP 2.—Sixty-seven cases in which the metastatic involvement of the central nervous system was a manifestation of general carcinomatosis, with metastatic lesions found in distant visceral organs and the skeleton as well as in the central nervous system.

GROUP 3.—Nineteen cases in which the metastatic involvement of the nervous system was a result of direct extension from a primary site contiguous with the nervous system, such as the nasal accessory sinuses, nasopharynx and scalp.

GROUP 4.—Twenty-two cases in which the metastatic involvement of the nervous system was limited to the spinal meninges.

In this study only the cases in group 1, in which the metastatic lesion simulated a primary intracerebral lesion, are considered. The other groups will be treated in greater detail in future presentations.

From the Department of Neurology and the Laboratory of Neuropathology, Philadelphia General Hospital.

1. Globus, J. H.: Metastatic Brain Tumor with Bronchogenic Carcinoma as the Primary Source, J. Mt. Sinai Hosp. **12**:533-564 (Jan.-Feb.) 1944.

In my survey of the pertinent literature, I have found no emphasis on the fact that the discrete metastatic lesions to the brain may be the only metastatic focus from a visceral organ.

INCIDENCE

In a series of approximately 6,000 autopsies of the nervous system performed in the last twenty years, metastatic involvement of the central nervous system was observed in 150, an incidence of 2.5 per cent of all autopsies on the brain. In the same series, the number of primary intracerebral tumors was (approximately) 300, and that of tuberculomas, 100. The incidence of metastatic lesions of the nervous system in relation to the total number of all neoplastic and granulomatous tumors of the nervous system therefore was 27.2 per cent. This incidence is higher than that reported by other investigators. In Cushing's Brigham Hospital series[2] metastatic tumors represented 4.2 per cent of all intracranial tumors. Elkington,[3] in a series of 805 cases of cerebral tumors studied at Queens Square Hospital, found the incidence of metastatic lesions to be approximately 9 per cent but commented that if patients with malignant disease who died at home or in special institutions were to be included the total incidence would be near 20 per cent. All these studies, being based on cases from neurologic or neurosurgical hospitals, are undoubtedly low in relation to the true incidence, since patients with frank malignant growths are not usually admitted to such institutions.

In a series of cases from general hospitals in which autopsies were performed the incidence of metastatic intracranial tumor is higher. Globus and Meltzer[4] reported an incidence of 13.5 per cent from the Mount Sinai Hospital; Baker,[5] an incidence of 17.9 per cent in a series at the University of Minnesota, and Courville,[6] an incidence of 26.9 per cent in a series at the Los Angeles County General Hospital. These statistics are more comparable to the figures for the present series, drawn from the Philadelphia General Hospital, an institution which, in addition to its general and neurologic wards, maintains a service for neoplastic diseases.

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2. Cushing, H.: Intracranial Tumors, Springfield, Ill., Charles C Thomas, Publisher, 1932, p. 8.

3. Elkington, J. St. C.: Metastatic Tumors of the Brain, Proc. Roy. Soc. Med. **28**:1080 (June) 1933.

4. Globus, J. H., and Meltzer, T.: Metastatic Tumors of the Brain, Arch. Neurol. & Psychiat. **48**:163 (Aug.) 1942.

5. Baker, A. B.: Metastatic Tumors of the Nervous System, Arch. Path. **34**:495-537 (Sept.) 1942.

6. Courville, C. H.: Pathology of the Central Nervous System: A Study Based upon a Survey of Lesions Found in a Series of Thirty Thousand Autopsies, Mountain View, Calif., Pacific Press Publishing Association, 1945, p. 351.

CLINICAL FEATURES

In only 19 of the 42 cases was the metastatic nature of the intracerebral lesion recognized clinically. In the other 23 cases the clinical diagnosis was that of a primary intracranial disorder, primary tumor being suspected in 15 cases, cerebral vascular accident in 7 cases, and Korsakoff's psychosis in 1 case.

Age.—The majority of patients were over 40 years of age, the youngest being 34, and the oldest 79. The age incidence is as follows:

Age, yr.	No. of cases
30-39	2
40-49	15
50-59	16
60-69	7
70-79	1
Unknown	1

Sex.—Men predominated in a proportion of 2.5:1, there being 30 men and only 12 women. This is a significant statistical difference, since in the total number of autopsies of the brain at the Philadelphia General Hospital the proportion of male and female patients was approximately equal. The higher incidence in male patients has been noted by other investigators—Globus and Meltzer[4] and Hare and Schwarz.[7] The reason for this difference is not clear. It has been suggested that the series previously reported may have been obtained from institutions which cared for few patients with malignant growths in the breast. This explanation is scarcely tenable in the present study, since at the Philadelphia General Hospital numerous cases of cancer of the breast come to autopsy. If intracerebral metastases were present, the incidence would be reflected in this series. Possibly the tendency of bronchogenic carcinoma to occur more frequently in males[8] may account for the disproportionate sex ratio.

Mode of Onset of Symptoms Referable to the Central Nervous System.—In only 7 cases was the onset of neurologic symptoms sudden or abrupt. In the great majority of cases, 31, it was slow and progressive. In 4 cases, the clinical history was vague with reference to this aspect, and the mode of onset must be considered indefinite.

Presenting Symptoms.—There was no characteristic neurologic symptom or sign occurring as the initial evidence of metastatic involvement of the nervous system. Headache was the most frequent initial symptom, being present in 12 cases (in 5 it was associated with vomiting, and in 7 it occurred alone). Mental symptoms were the first manifestation in 10 cases. The commonest initial sign was hemiplegia, which was found in 10 cases. Generalized convulsions initiated the onset in 6 cases; jacksonian seizures in 1. In 1 case pain in the face was the first complaint.

7. Hare, C. C., and Schwarz, G. A.: Intracerebral Carcinomatous Metastases, Arch. Int. Med. **64**:542-565 (Sept.) 1939.

8. Klemperer, P., cited by Globus and Meltzer.[4]

Symptoms.—Most of the symptoms and signs encountered were those indicative of focal neurologic disorder, mental disturbance or increased intracranial pressure.

Focal Neurologic Signs: Focal neurologic signs were present in 31 cases (74 per cent). In 6 cases they occurred alone, and in the same number of cases they were associated with evidence of increased intracranial pressure. In 8 cases both focal signs and mental symptoms were present, while in 11 cases focal signs, mental disturbances and signs of increased intracranial pressure were all present.

Hemiplegia or hemiparesis was the most frequently encountered focal sign, being present in 22 cases. In 5 cases it was accompanied with aphasia, and in 2 cases, with hemianesthesia. Expressive aphasia without hemiplegia was present in 1 case and paraplegia in another. Cerebellar ataxia occurred in 2 cases. Pseudobulbar phenomena and dysarthria were each present in 1 case.

Mental Symptoms: Mental symptoms occurred frequently, being present in 26 cases (62 per cent). In 3 cases they represented the only sign of involvement of the nervous system occurring without any concomitant neurologic sign. In these cases, and in those in which mental symptoms were the initial manifestations, appearing prior to the onset of neurologic signs (10 cases), the true nature of the disorder was not recognized until later; and many of the patients were first admitted to the psychopathic service.

In 23 cases focal signs and/or signs of increased intracranial pressure were associated with the mental disturbances.

The prevalence of mental symptoms associated with metastatic tumors of the brain has been noted by other investigators. Grant[9] found dispositional changes and dull mentality in 13 of 49 cases and cautioned that if a woman of middle age suddenly exhibits a psychosis with asthenia and signs of intracranial pressure a primary focus of cancer from which cerebral metastasis may have occurred should be mose carefully sought.

In our series, no specific psychiatric syndrome was found. Disturbances in consciousness, confusion and disorientation were most frequent. The mental symptoms encountered were as follows:

Mental Symptoms	No. of Cases
Disorientation	7
Confusion	7
Mental dulness or lethargy	6
Stupor	6
Disturbances of memory	4
Negativism	3
Coma	2
Illusions	1
Hallucinations	1
Delusions	1
Personality changes	1
Violence and noisiness	1
Unspecified	1

9. Grant, F. C.: Concerning Intracranial Malignant Metastases, Their Frequency and Value of Surgery in Their Treatment, Ann. Surg. **84**:635-646 (Nov.) 1926.

Increased Intracranial Pressure: Evidence of increased intracranial pressure was present in 24 cases (60 per cent) and absent in 10 cases. In 6 additional cases subjective symptoms such as headache and vomiting, suggestive of increased intracranial pressure were present, but objective evidence of papilledema, roentgenologic changes or increased spinal fluid pressure was lacking. In 2 cases the records were incomplete with respect to the presence or absence of increased intracranial pressure.

Papilledema was found in 16 cases, and the spinal fluid pressure was elevated in 13 of the 16 cases in which lumbar puncture was made. Roentgenologic changes suggestive of increased intracranial pressure were present in 7 cases.

Changes in the composition of the cerebrospinal fluid were not common except for increase in protein—average 158 mg. per hundred cubic centimeters. In 2 cases there was slight pleocytosis, but in both cases the serologic reactions of the spinal fluid were also positive and the cellular changes were best explained on the basis of the concomitant syphilis of the central nervous system.

Other Neurologic Signs and Symptoms.—Except for pupillary disturbances, which were present in 11 cases, and strabismus, present in 1 case, signs most likely attributable to increased intracranial pressure, evidence of involvement of the cranial nerves was uncommon. In 1 case pain over the eye was complained of, and in the other, transient deafness; but in both cases objective supporting signs were lacking.

Generalized convulsions occurred in 10 cases, and jacksonian seizures, in 1 case. Incontinence was present in 3 cases and proptosis in 1 case.

Extraneural Symptoms.—General somatic signs which might have suggested the presence of a primary neoplasm, such as asthenia, cachexia, anemia and loss of weight, were present in 18 cases (42 per cent). In 12 of the cases in which symptoms of wasting were conspicuous the metastatic nature of the lesion was recognized during life, but in 6 cases these symptoms were so slight that their significance was overlooked and the true metastatic nature of the intracerebral tumor was recognized only at autopsy. The loss in weight ranged in individual cases from a few to 40 pounds (18.1 Kg.), the average being 10 pounds (4.5 Kg.). Severe anemia rarely was observed. In the remaining 24 cases loss of weight, anemia and cachexia were not conspicious.

In this series roentgenograms of the chest were not made routinely. Of the group in which the metastatic nature of the lesion was recognized clinically, roentgenograms of the chest were made in 11 cases, and showed evidence of neoplasm in only 7. In cases in which the metastatic nature was not appreciated until autopsy, the roentgenograms of the chest showed no lesions in the 7 cases in which they were made.

Clinical signs indicative of a pulmonary lesion were present in 11 cases. The history of previous amputation of a breast because of suspected malignant growth, the findings on pelvic examination and the presence of a tumor of the parotid gland assisted in suggesting the metastatic nature of the intracerebral lesion in other cases.

Duration of Illness.—The duration of life from onset of symptoms to death ranged from one day to two years, the average duration being approximately four months.

Surgical Intervention.—Craniotomy was performed in 4 cases in which the metastatic nature of the lesion was unsuspected, and in 2 other cases operation was advised but refused. The maximum period of survival following operation was two months. In 1 case in which the metastatic nature of the lesion was appreciated clinically a decompression was done for relief of severe headache.

PATHOLOGIC OBSERVATIONS

Location of Primary Neoplasm.—At autopsy the primary neoplasm was most frequently encountered in either the bronchus or the lung—21 cases (50 per cent). In 9 cases no primary source could be definitely ascertained at autopsy, although the histologic picture of the intracranial lesion left no doubt as to the metastatic nature of the intracranial tumor. In 2 cases the primary tumor was observed to be neurogenic, arising in the adrenal medulla, and in 2 cases, it was sarcomatous. In the remaining cases the primary lesion was carcinomatous. From the clinical standpoint, it is of interest that in all cases in which the primary neoplasm was neurogenic or sarcomatous the onset was abrupt. This, together with the cryptic location and relative asymptomatic nature of the primary lesion, masked the true neoplastic nature of the process and led to an erroneous diagnosis of a cerebral vascular lesion.

The location and frequency of the primary neoplasms in this series were as follows:

Location	No. of cases
Bronchus or lung	21
Breast	3
Primary neuroblastoma of the adrenal gland	2
Cervix uteri	1
Ovary	1
Stomach? (autopsy limited to brain and cord)	1
Liver	1
Lymphosacroma of mediastinum	1
Sarcoma of spleen	1
Mixed tumor of the parotid gland	1
Primary site undetermined	9

Except for involvement of the regional lymph nodes, there were no extraneural metastases in any of the cases included in this series. Cases in which metastases involved other extraneural organs were considered as showing generalized carcinomatosis and are not included in this series.

Location of Metastatic Lesions in the Nervous System.—At autopsy, the metastatic tumors were observed throughout the various areas of the brain, and there was nothing to suggest predilection for any particular locus. Of interest was the comparatively rare involvement of the cerebellum. In only 1 case was the cerebellum the site of a solitary nodule, and in only 5 instances was it part of a multiple metastatic involvement. Globus and Meltzer,[4] likewise, found the cerebellum involved in only 2 of their 57 cases. Baker[5], and Hare and Schwarz,[7], however, reported a high incidence of metastatic involvement of the cerebellum.

The location of the tumors within the various areas of the brain is tabulated as follows:

Locus	Solitary Involvement	Site in Multiple Involvement	Total No. of Cases
Cerebrum	18	30	48
Cortex and subcortex			
Frontal lobe	7	9	16
Parietal lobe	3	5	8
Frontoparietal cortex	1	1	2
Paracentral lobule	1	0	1
Rolandic area	2	3	5
Temporal lobe	3	1	4
Occipital lobe	0	5	5
Cortex—unspecified	0	2	2
Area of internal capsule and basal ganglia	1	4	5
Cerebellum	1	5	6
Pons	0	3	3
Diencephalon	0	2	2
Pituitary gland	0	1	1
Spinal meninges	0	1	1
Cortical meninges (by direct extension from underlying cortical area)	0	5	5

Krasting[10] and Kikuth[11] claimed that the left cortex was more frequently involved than the right, but the significance of this observation was questioned by Baker[5]. In the present series, the left side of the brain alone was involved in 19 cases and the right side alone in 12, while in 8 cases both sides were involved.

Multiplicity of Metastatic Lesions.—In 17 cases there was a single metastatic nodule in the nervous system, while multiple metastatic foci were present in 25 cases. The maximum number of metastatic tumors observed in a single case was 5.

General Pathologic Consideration.—The metastatic tumors encountered in this series were discrete, isolated nodules, usually solid but occasionally cystic, ranging in size from 1 to 9 cm. in diameter and occurring either deep within the white matter or superficially in the cortex. They were limited to the neural parenchyma except in 5 cases in which the contiguous dura was involved. In no case in this series was there widespread carcinomatous involvement of the meninges, such as

10. Krasting, K.: Beitrag zur Statistik und Kasuitstik metastatischer Tumoren, besonders der Carcinommetastasen im Zentralnervensystem (auf Grund von 12,730 Sektionen der pathologisch-anatomischen Anstalt Basel), Ztschr. f. Krebsforsch. **4**:315-379, 1906.

11. Kikuth, W.: Ueber Lugencarcinom, Virchows Arch. f. path. Anat. **255**:107-128, 1925.

was originally described by Saenger[12] and has since been reported by numerous authors under the descriptive term "carcinomatosis of the meninges" (Hassin[13]). In 1 case in which the metastatic lesion appeared to arise from the floor of the third ventricle in the region of the ventral hypothalamic nuclei, there was direct extension into the optic chiasm, and sections of the optic nerves showed a central core of tumor. The pituitary gland was also involved by way of the infundibular stalk, the anterior lobe being almost completely replaced with tumor tissue. The metastatic nodules were observed in both the cortical gray and the white matter. There was no special predilection for their localization predominantly in the cortical gray matter or at the junction of the gray and the white matter, as claimed by Rich[14] and Elkington.[3]

COMMENT

It is again emphasized that in the 42 cases reported in this series the intracerebral metastases were the only metastatic lesions observed at autopsy. Why the intracerebral metastases should be the only metastatic foci present is not clear. Although the most generally held theory is that metastases to the nervous system takes place secondarily from the lungs by way of the blood stream, solitary metastatic involvement of the brain without accompanying metastatic seeding of the lungs and other viscera may also be explained on the basis of the rich anastomosis existing between the epidural and vertebral veins and the veins of the thoracoabdominal cavity. This was demonstrated by Batson,[15] who stated the opinion that compression of the trunk, such as that produced by straining, coughing and lifting, raises the truncal pressure sufficiently to produce a retrograde flow of blood into the vertebral venous system, whereby tumor cells could be driven from the abdominal veins into the vertebral veins and then into the cranial cavity without going through the lung.

Failure to appreciate that a malignant tumor of an extraneural organ may give rise to metastases limited exclusively to the brain contributes to the difficulty in many cases of recognizing the metastatic nature of such lesions. Especially is this true when the primary neoplasm is so minute, so situated or otherwise of such a nature that it gives rise to no detectable clinical or roentgenographic symptoms. In such cases a cor-

12. Saenger, A.: Ueber Hirnsymptome bei Carcinomatose, Neurol. Centralbl. **19**:187-192 (Feb.) 1900.

13. Hassin, G. B.: Histopathology of Carcinoma of the Cerebral Meninges, Arch. Neurol. & Psychiat. **1**:705 (June) 1919.

14. Rich, G. J.: The Distribution of Metastatic Tumors in the Cerebrum, Arch. Neurol. & Psychiat. **23**:742-749 (April) 1930.

15. Batson, O. V.: The Function of the Vertebral Veins and Their Role in the Spread of Metastases, Ann. Surg. **112**:138-149 (July) 1940.

rect clinical diagnosis of the metastatic nature of the intracranial tumor is practically impossible. Since a high proportion of metastatic intracranial lesions are secondary to a primary malignant tumor in the bronchus or lung, it was suggested by King and Ford[16] that a roentgenographic examination of the lungs should be made routinely of all patients 40 years of age or older who present signs and symptoms suggestive of an expanding intracranial lesion. This suggestion should be regarded as mandatory, but in view of the pathologic observations in this series it is obvious that even the most careful and detailed roentgenographic study of the chest will often fail to reveal the presence of a primary tumor of the lung. In some om these cases it required a prolonged and diligent search post mortem to reveal a minute primary focus. In others no primary extraneural tumor could be identified. It is hardly conceivable that such inconspicuous lesions can be disclosed by roentgenographic examination. Examination of the lungs contributes nothing toward establishing a correct diagnosis if the primary tumor arises in a locus outside the lungs and the only metastatic foci are intracerebral. It seems likely, therefore, that many discrete metastatic tumors of the brain will be mistaken for primary intracranial tumors until histologic examination of the tissue is possible, either at biopsy or at autopsy. Such was true in 4 cases in this series in which operation was performed.

As an aid to a more accurate clinical diagnosis of intracranial metastatic tumor, some authors have attempted to formulate certain constellations of signs and symptoms which should arouse suspicion of the metastatic nature of the intracranial tumor. Ferguson and Rees[17] and Elkington[3] stressed the diagnostic significance of the occurrence in a middle-aged patient of the acute onset of headache and intracranial symptoms accompanied with severe wasting. Globus and Selinsky[18] emphasized that the acute onset of cerebral symptoms followed rapidly by the development of disseminated and rapidly progressive neurologic signs, together with the occurrence of symptoms of increased intracranial pressure, except for papilledema, which may be absent, should suggest a metastatic neoplastic process. Such a probability is strengthened further by the appearance of progressive wasting and asthenia to a degree out of proportion to that usually encountered with primary tumors of the brain.

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16. King, A. B., and Ford, F. R.: A Clinical and Anatomical Study of Neurological Conditions Resulting from Metastases in the Central Nervous System Due to Carcinoma of the Lung, Bull. Johns Hopkins Hosp. **70**:124-156 (Feb.) 1942.

17. Ferguson, F. R., and Rees, W. E.: Cerebro-Spinal Metastases from Unsuspected Pulmonary Carcinoma, Lancet **1**:738-744 (April 5) 1930.

18. Globus, J. H., and Selinsky, H.: Metastatic Tumors of the Brain, Arch. Neurol. & Psychiat. **17**:481-513 (April) 1927.

The observations in the cases included in this series raise doubts concerning the value of such diagnostic complexes, and in my opinion metastatic tumors of the brain do not present any specific sympton complex which would permit a ready differentiation clinically from primary intracranial neoplasms, such as spongioblastoma multiforme or other focal lesions. The onset may be abrupt or slowly progressive. Wasting, cachexia and asthenia were not conspicuous in many cases in this series, and frequently were absent. When present, they should serve to arouse suspicion concerning the metastatic value of the intracerebral lesion, but their absence by no means lessens the possibility of the intracerebral lesion being metastatic. Since metastatic lesions show no particular predilection for any specific area of the brain, one would not expect any uniformity of focal signs, and the presence of signs indicative of increased intracranial pressure would be contingent on various factors, such as the location of the tumor in respect to impingement on the ventriculosubarachnoid pathways, venous outflow, size, rapidity of growth and compression or destruction of neural tissue. In my opinion, only when intracranial manifestations occur in association with substantial clinical or roentenographic evidence of a primary extraneural neoplastic focus can the diagnosis of the metastatic nature of the intracranial lesion be reasonably substantiated clinically. In the absence of evidence of a primary focus, the metastatic nature of the intracranial lesion usually is not suspected prior to operation or autopsy. However, because of the frequency with which metastatic intracranial tumors occur, the possibility that any intracranial lesion may be metastatic in origin should be seriously considered, particularly if the patient is in the older age group, and a diligent and careful physical and roentgenographic examination, including anteroposterior and lateral views, should be made in an attempt to determine the presence of a primary neoplastic focus. Failure to find a primary focus clinically does not, however, eliminate the possibility that the intracerebral lesion may be metastatic.

Although Hare and Schwarz[7] stated their opinion that patients with metastatic intracerebral tumors do not tolerate surgical procedures well, and the duration of life is shorter in patients who have been operated on, surgical intervention would appear to be indicated unless the metastatic nature of the intracerebral lesion is established beyond doubt. Even when the metastatic nature of the lesion is recognized, surgical intervention may be justified as a palliative measure for relief of intractable headache and other severe symptoms of increased intracranial pressure.

SUMMARY

Metastatic tumors comprised 27.2 per cent of neoplasms of the nervous system observed in a series of 6,000 autopsies of the brain performed at

the Philadelphia General Hospital. The high incidence of metastatic lesions is attributed to the fact that the autopsy material was derived from all services in a large general hospital.

In 42 cases a discrete intracerebral metastatic tumor was the only metastatic focus observed at autopsy, and the clinical symptoms simulated those of a primary intracerebral lesion.

In many cases the primary neoplasm is extremely small and is observed at autopsy only after careful search; in others a primary tumor cannot be identified even after a careful autopsy. In such cases a correct clinical diagnosis of the metastatic nature of the intracerebral lesion is impossible.

Carcinoma of the bronchus or lungs is the most frequent primary source of a discrete intracerebral metastatic tumor. Carcinoma of the breast, uterus, ovary and liver, as well as neurogenic tumors of the adrenal gland may give rise to similar metastases.

There are no characteristic clinical symptoms distinguishing intracerebral metastatic tumor. The onset may be abrupt or slow. Focal neurologic signs are usually present and may be associated with mental disturbance. In a few cases mental disturbance alone occurs. Evidence of increased intracranial pressure may or may not be present. Cachexia and wasting are often inconspicuous or absent.

The intracerebral metastatic lesions may be solitary or multiple and occur in all areas of the cortex, cerebellum and brain stem.

A correct clinical diagnosis of the metastatic nature of an intracerebral lesion is difficult unless the primary neoplasm gives rise to symptoms. A careful, detailed roentgenographic examination of the chest should be made in all persons 40 years of age or older who present evidence of an intracerebral lesion, and such a study may be of diagnostic help if the primary tumor is in the bronchus or lung. A negative roentgenogram of the chest does not preclude the possibility of the intracerebral lesion being metastatic, since the primary tumor, if in the lung, may be too minute to be detected in a roentgenogram. A roentgenogram of the chest is of no diagnostic aid when a primary tumor located elsewhere in the body gives rise to metastases located only within the brain.

133 South Thirty-Sixth Street (4).

PROGNOSTIC SIGNIFICANCE OF INCIDENTAL ACUTE SOMATIC DISORDERS IN THE COURSE OF PSYCHOSES

Reevaluation of Five Cases Fourteen Years Later

ALFRED GORDON, M.D.
PHILADELPHIA

INFECTIOUS diseases may at any period of their evolution create psychotic disorders of various characters. But they may also have a favorable influence, even of years' duration, on preexisting mental disorders. It is the latter aspect of the relationship that will be considered here.

As far back as 1844 Jacobi,[1] and in the next few years von Rienecker, Griesinger, Neumann, Kraft-Ebing, Régis, Kraepelin, Fielder[2] and others, reported a satisfactory influence of incidental disease on the course of the original psychosis.

The psychotic disorders which have thus been benefited are of wide variety; mental depression; hyperkinetic and expansive states of manic-depressive psychosis; persecutory delusions of the paranoid type; delusive and hallucinatory states of dementia precox; agitated states of involutional melancholia, and psychasthenic episodes. Of the incidental disorders influencing the original psychoses, the following have been recorded: exanthematous diseases, acute inflammatory rheumatism, suppurative conditions and trauma to the cranium. As to the degree of improvement in the psychoses, various statements have been recorded, the effects ranging from some amelioration or from remission to complete recovery, and this was true not only of the acute psychoses and of mental disorders of recent date but of chronic psychoses of long or of very long standing, such as dementia precox or paranoid states, for which an unfavorable prognosis was admitted.

It is observed that the agitation and anxiety of melancholia agitata gradually subside and finally disappear; the confusion, disorientation, negativism and stereotyped attitudes are replaced by normal behavior; the affective and emotional indifference and indolence of dementia precox become much less profound and in some cases entirely disappear,

1. Jacobi, K. W.: Die Hauptformen der Sedenstörungen in ihren Beziehungen zur Heilkunde noch der Beobachtung geschildert, Leipzig, Weidmann, 1844.

2. Fielder: Allg. Ztschr. f. Psychiat. **43**:208, 1886-1887.

and the tension of psychomotor inhibition of schizophrenia is greatly diminshed, so that the patient gains a better grasp of his surroundings. As to whether the effect is only a temporary remission, as Weygand saw it, or a more prolonged improvement, the records differ. Improvement is also seen in a group of unclassified cases with a great variety of psychotic phenomena, and in some cases there is even a return of certain psychic functions which have lost their activity. In manic states the expansion and excitement not only subside, but in some cases entirely disappear. Hallucinatory phenomena in the course of various mental disturbances likewise improve or disappear.

Since the records in the literature show a variability in the influence of an incidental disease on the course of the original psychosis, any additional observation must be of practical value, especially when the question of prognostic significance arises. An unusual opportunity presented itself for a possible solution of the latter.

In the 5 cases about to be described, I was confronted with an extremely acute onset of the incidental disorder, a circumstance which may be regarded as comparable to the therapeutic sudden shock extrinsically induced with electric current, insulin or "metrazol." The only difference is that, instead of an external agent, the shock in my 5 cases was induced by internal, or metabolic, agents. This highly interesting parallel becomes significant from the point of view of predicting with greater probability of correctness the outcome of the original psychosis, which was disoriented, and finally eliminated, by a chemical attack within the organism. A fourteen year interval without a trace of the return of the original psychotic episodes, which prior to the "internal shock" recurred for years, is instructive not only for a better understanding of the mechanism of shock therapy of psychoses but for practical prediction as to the outcome of the psychosis.

REPORT OF CASES

Case 1.—N. F., a man aged 39, at the age of 13 began to show peculiarities of behavior, namely, hostility and aggressiveness toward his mother, for whom he had always exhibited great affection. With it he lost interest in his surroundings, his school friends, his brothers and sisters and his school work. At the least controversy, he would become very talkative and use profane language. Gradually, this condition would subside, and he then entered a state of self isolation. He avoided people, even his parents and relatives, and for days he was moody, irritable and silent. These periods of alternating mood occurred every few months. However, a searching history of the patient's entire life revealed the appearance of abnormalities at a still earlier age. As a child he was extremely shy and could not tolerate the company of other children. He was irritable, always discontented and never smiled. At no time could his family physician find any disturbed function which required attention. His school work was only mediocre. He never showed any special desire to accomplish anything in the in-

tellectual sphere. The episodes which appeared when he was 13 years old continued intermittently until the age of 27, when he had lobar pneumonia. He had a high temperature, of from 102 to 105 F., and his illness, which was severe, lasted five weeks.

The following record is highly interesting: The parents stated that at the height of his fever (temperature, 105 F.) he became delirious. Four days later the delirium disappeared, and his personality traits underwent a radical change. He became amiable; asked repeatedly for his parents, brothers and sisters; insisted on their remaining with him for hours, enjoying their company and that of his former boy friends; spoke of his plans for the future, and expressed a strong wish to join his father in the real estate business. When he finally regained his health, his behavior, relation to his surroundings, judgment and views on events and life in general assumed an apparently normal aspect. For the subsequent fourteen years there has been no evidence of a return of his condition which was so striking prior to his pneumonia. At present he does more or less satisfactory work with his father. He has never married.

The adjustment in his case was of only a social character. The patient belongs fundamentally to the schizoid type. His entire adolescent and adult life bears strong evidence of schizophrenic tendencies.

Case 2.—B. C., a woman aged 42, presented from the age of 17, during a period of eleven years, fluctuating phases of extreme exaltation, followed by periods of extreme depression. In the latter there were at times pronounced stupor and motor immobility, with catatonic attitudes. Resistiveness, with a tendency to carry out orders diametrically opposed in meaning to those requested, was striking. When she was told to flex her head, she extended it; when told to open her mouth, she closed it tightly; when told to lean forward, she leaned backward. This antagonistic motor reaction, combined with the general immobility, was so persistent that the diagnosis of catatonia of dementia precox had been considered by several psychiatrists. Recurrences of both states took place every few months. During the last attack of extreme exaltation, she was exposed for many hours to cold, being scantily clad for a dance. Bronchopneumonia developed. Her temperature was very high for many days, and she appeared extremely ill. The exalted state, during which period the somatic disease developed, promptly disappeared. Complete somatic recovery followed, but only after a prolonged convalescence. The original psychotic manifestations, which were so striking between the ages of 17 and 29, have not recurred during the last fourteen years.

Case 3.—D. O'B., a man aged 50, had before his marriage, at the age of 27, had three attacks of typical depressive psychosis, each lasting three to six weeks. The usual symptoms were presented: self isolation, hopelessness, with occasional attempts at self destruction, and total loss of interest. Three similar attacks occurred during the first five years of his married life. The last attack had an unusually long duration, during which he had an illness diagnosed as influenza. He was bedridden for many weeks, with a temperature fluctuating from 102 to 105 F. Meningeal symptoms were observed. After several months of this fluctuating pathologic state, followed by a protracted convalescence, he recovered. Since then, during a period of almost fifteen years, there has been no recurrence of the depressive psychosis.

Case 4.—F. N., a man aged 36, had been considered peculiar from childhood, according to his relatives who took charge of him as an orphan. The father was alcoholic, and the mother spent several years in a psychiatric institution. The

patient had outbursts of extreme anger, violence and incendiary tendencies. Between the ages of 10 and 20 he had cyclothymic periods, lasting from a few hours or days to two months. At the age of 20, there was an unusually long and pronounced phase of depression, accompanied with a vague delusional attitude toward the relatives with whom he continued to live. Once he walked away from home in the middle of the night and was attacked by a bandit. He was picked up on the street, bleeding profusely from the mouth and urethra. Loss of consciousness was not complete. At his direction, he was brought home. The bleeding continued two days but finally subsided, and disappeared on the fifth day. During his bedridden condition the patient was able to give a clear account of the occurrence, and the depression and delusional attitude totally disappeared. He made an eventual recovery. With it, there was a radical change in the personality. He became amiable, attentive and respectful toward his relatives. The social adjustment seemed to be highly satisfactory. For fourteen years he had been employed and has kept his job of assistant jeweler. The former manic-depressive episodes have not recurred.

Case 5.—K. R., a single woman aged 34, had had periods of manic-depressive psychosis from the age of 14, the episodes occurring immediately before the onset of her menses and lasting during, and a few days after the cessation of, the flow. In addition to the hyperkinetic state, the manic phases presented unusually expansive ideas in the direction of her accomplishments. The last psychotic attack, while also occurring at the menstrual period, was especially prolonged, lasting three months, with emphasis on the depressive phase. Being careless with regard to covering in cold days, she became ill with influenza and, later, a pulmonary infection, with a temperature of 101 to 104 F. The temperature remained high for six days, and during that time a decided change was observed in her psychotic condition. The depression gradually disappeared. She became more accessible and manageable. She was able to criticize reasonably her surroundings and expressed surprise at her former expansive ideas concerning her personal talents, although she knew that her knowledge of music was only mediocre. The febrile state was protracted, but she finally recovered. Since then, for fourteen years, she has been free from psychotic attacks.

COMMENT

The 5 cases just described offer conclusive evidence of a beneficial effect of the incidental intervention of an acute somatic disorder during the course of a psychosis, even of long standing. They raise several questions. What was introduced into the economy of the psychotic patient by the acute somatic disorder? Why should the new disorder be antagonistic to the preexisting psychosis? Of what does this antagonism consist? In the cases of a febrile infectious process, one has more solid ground for interpretation than in the cases of afebrile illness.

In the cases of a febrile illness one is warranted in speaking of a modification of the chemical constitution of the organism by elaboration of new material which may be antagonistic to the chemistry of the psychotic organism. Such a possibility is admissible in some psychoses. In dementia paralytica, alcoholic intoxication, chronic lead poisoning and uremia, in all of which one encounters mental disorders, such as disorientation, de-

pression or euphoria and delirious and hallucinatory states, the morbid chemistry of the organism is etiologically responsible for these mental disturbances, and with removal of the noxious substance the latter disappear. Unfortunately not all fully developed psychoses can be explained on such evident chemical grounds. The 5 cases described here present no evidence for interpretation of the psychotic disorders on a well established chemical basis. It is difficult, if not impossible, to rely on the explanation of the sudden formation of antagonistic chemical material in cases of manic-depressive or schizophrenic outbursts interrupted in their course by shock, whether from trauma or from profuse hemorrhage (case 4), so as to arrest a morbid psychic process. The pathogenesis of psychoses in general, in the present state of knowledge, is only speculative. It is supposed that psychotic disturbances are the result of affective conflicts, with loss of conscious control of the affective states. Since spontaneous affective adjustment is wanting, any new emotional element introduced by an incidental somatic disorder, whether it is a hemorrhage or trauma or a strong elevation of temperature, seems to act as a specific affective stimulus, which conditions the primary affective complex and, by readjustment of the elements of the latter, frees the personality from the disturbing conflict. The histories of the 2 schizoid patients in this series presented evidence of early schizophrenic trends, with loss of integration in various emotional fields and incongruous lack of inhibition in displaying certain emotions. When the incidental acute somatic disturbance intervened, radical changes were soon observable in the individual personality traits: Shyness, shut-in attitude, timidity, fluctuations of mood and, especially, the strong anxiety attacks, all greatly improved.

For fourteen years the 5 patients were seen at various intervals, about every six months, and their condition was reappraised, with emphasis on their affective responses to the environment and their adaptation. Although schizoid persons, by the very nature of their inherent makeup, remain as such through their life cycle, and are subject to schizophrenic attacks under various intrinsic or extrinsic unfavorable influences, these 5 cases demonstrate that organic deficits in such personalities may be amenable to repair by the breaking up of old configurations, by liberation and utilization of the affective side of the personality. During the quite uneventful period of fourteen years, one could witness an astonishing clinical restructuralization of the 5 personalities, with a very satisfactory adjustment to old and new environmental factors. These phenomena were apparently produced by reparatory responses to the intrinsic shocks induced by incidental acute somatic disorders, in the form of intense febrile states, trauma or sudden, abundant hemorrhage. Neither structural pathology nor chemistry has been able to give scientifically the

raison d'etre and the modus operandi of shock therapy in exogenous form, whether induced with the electric current, with insulin or with "metrazol," or in endogenous form, as in the 5 cases here described. Psychologic interpretation of the favorable influence of shock, in spite of its reasonableness and logicality, such as the phylogenetic fear of death and the consequent stimulation to recovery, is, again, highly speculative.

Despite the controversial aspects of the pathogenesis of psychiatric manifestations, one must admit that favorable results are sometimes observed from certain empiric methods of therapeutic approach. The present cases, in which the patients benefited greatly from an infectious febrile state, can be placed in parallel with the records in the literature which show that fever-inducing vaccines, bacterial cultures and toxins and proteins may give satisfactory results. It may be that in all such cases the increase in temperature alone may be the healing principle. Reactive depression, anxiety neurosis, the anxiety of melancholia and acute schizophrenic states have been benefited by all such therapeutic attempts. It seems, therefore, that shock, such as artificially induced fever or fever from an acute infectious process, as well as shock from trauma or abundant hemorrhages, may all be considered as endogenous shock, analogous to the exogenous shock induced with the electric current, "metrazol" or insulin.

The present 5 illustrative cases prove this contention. Moreover, they serve as a demonstration of the value of shock in the prognosis of psychoses. If fourteen years has passed without a single attack of malignant psychotic manifestations, after frequent outbursts for a number of years, and this effect is apparently due to the endogenous shock, the mental status of the patient stands a good chance of being maintained in the future, and the psychiatrist is in a favorable position to render an encouraging prognosis, even in cases of long duration.

SUMMARY

Five patients who had had intermittent psychotic attacks for many years underwent a remarkable arrest of the malignant manifestations, with no recurrence of the psychosis in any case for fourteen years after the last attack. The recovery was brought about in each case by an incidental somatic disorder, such as a high febrile state during pneumonia or influenzal meningitis, trauma or abundant hemorrhage.

What, in these circumstances, led to cessation of the psychotic disorders? What are the profound changes which produced the remarkable results in the affected personalities? Is metabolic shock to the nervous system, such as is observed in electroshock, "metrazol" shock or insulin shock, the factor involved? The modus operandi in both endogenous and exogenous shock is still unknown. All one can say is that there is a strik-

ing analogy between the two forms. The present 5 cases possess an instructive feature, not reported in the literature, namely, complete absence during fourteen years of observation of the former, frequently recurring psychotic episodes. The latter circumstance presents an important element in establishing a favorable prognosis of a psychosis.

The salient features in the histories of the 5 cases are presented, and the pathogenesis of the psychosis is discussed.

Clinical observation, accompanied, of necessity, with experimental investigations on a large scale, is indispensable to a clearer understanding of the intimate relation of somatic and psychotic phenomena.

A plea is made for a tolerant attitude toward the various views concerned with the pathogenesis of abnormal psychotic manifestations. Only by a judicial approach to the solution of this difficult problem, based on scentific data from any source, can one expect worth while therapeutic results.

1900 Locust Street.

Abstracts from Current Literature

Edited by Dr. Bernard J. Alpers

Physiology and Biochemistry

THE EFFECT UPON THE COLON OF ELECTRICAL STIMULATION OF FOREBRAIN AREAS IN CATS. E. H. INGERSOLL and LOUISE JONES, Am. J. Physiol. **146**: 187 (May) 1946.

Ingersoll and Jones stimulated subcortical areas of the forebrain in cats and observed the types of responses of the colon and the mode of transmission of the impulses to the colon. A delayed excitatory response was most frequently encountered. This was elicited from the hypothalamus and other subcortical structures. Section of the cord at the sixth lumbar vertebra abolished or decreased the response. The immediate excitatory response was elicited at all levels of the hypothalamus and at most levels of the thalamus and telencephalon. Section of the cord abolished or greatly decreased the response. The inhibitory response was immediate and was obtained from any point in the hypothalamus, as well as other areas studied. The authors state that their experiments offer no evidence for delimited sympathetic or parasympathetic areas in the hypothalamus.

FORSTER, Philadelphia.

THE BULBAR INHIBITORY MECHANISMS IN CONCUSSION. R. RHINES, H. W. MAGOUN and W. F. WINDLE, Am. J. Physiol. **146**:344 (June) 1946.

Rhines, Magoun and Windle studied the effects of cerebral concussion on the patellar reflex in cats anesthetized with chloralosane (a compound of chloral and dextrose). At the time of the concussive blow some motor discharge occurred. Brief impairment of the patellar reflex was followed by longer-lasting hyperreflexia. This reflex augmentation was paralleled by a reversible depression of the excitability of the bulbar inhibitory mechanism of the medulla, and this suggests that the alterations in the patellar reflex may be due to a release from the inhibitory mechanism of the bulb.

FORSTER, Philadelphia.

OBSERVATIONS ON THE INTERACTION OF NERVE IMPULSES IN THE GRAY MATTER AND ON THE NATURE OF CENTRAL INHIBITION. BIRDSEY RENSHAW, Am. J. Physiol. **146**:443 (June) 1946.

Renshaw points out that stimulation of the sixth lumbar dorsal root produces a contraction of the quadriceps muscle after 1 to 1.1 millisecond. This discharge can be inhibited by stimulating the seventh lumbar dorsal root 1 to 1.1 millisecond before the motoneuron discharge. Increasing the time interval between the two volleys increases the inhibition until it becomes complete. When the potential changes of the motoneurons are determined, they are found to be altered by the inhibiting stimulus, and this is considered evidence that during the synaptic delay interaction occurs between impulses in the terminal branches of the two groups of dorsal root fibers. Antidromically evoked somatic action potentials are not

altered by the inhibiting stimulus, thus further indicating that the direct inhibition is in part at least between the two groups of premotor fibers.

FORSTER, Philadelphia.

CIRCULATORY EFFECTS OF PARTIAL CEREBRAL ISCHEMIA. DAVID F. OPDYKE, Am. J. Physiol. **146**:467 (June) 1946.

Opdyke studied the effects of occluding the inhominate and left subclavian arteries in dogs. Because of collateral circulation, an effective cerebral ischemia did not occur unless the carotid back pressure was reduced to 30 mm. of mercury or less; if the pressure was greater than this, reflex movements were not abolished. Therefore partial cerebral ischemia cannot be used as a substitute for anesthesia in studying the dynamics of hemorrhagic shock.

When the carotid back pressure fell below 30 mm. of mercury circulatory failure occurred in some animals but not in others. In the animals without failure an increased peripheral runoff time was demonstrated, and this was presumably due to increased peripheral resistance.

Circulatory failure developed on the restoration of cerebral circulation in those dogs which had maintained a normal blood picture during the ischemic period.

FORSTER, Philadelphia.

CUTANEOUS DISTRIBUTION OF PERIPHERAL NERVES IN RHESUS MONKEYS AS DETERMINED BY THE ELECTRICAL SKIN RESISTANCE METHOD. S. R. BRUESCH and CURT P. RICHTER, Bull. John Hopkins Hosp. **78**:235 (April) 1946.

The observations reported concern the immediate and certain of the prolonged effects produced on the electrical resistance of the skin by transections of peripheral nerves in rhesus monkeys. Such transections of peripheral nerves produce nervousness and sweating because postganglionic sympathetic nerve fibers are severed, as well as afferent and somatic efferent fibers.

Operations were performed on 2 rhesus monkeys (Macaca mulatta) under ether anesthesia. A peripheral nerve in an extremity was exposed at a selected level, transected and sutured. Rotation of the cut ends at the time of suture was minimized by the placing of sutures in corresponding surfaces of the nerve proximal and distal to the level of proposed transection before the cut was made. After the transection, the cut ends were sutured with black silk swedged onto fine round needles. Connective tissues and skin were sutured with heavier black silk. No dressings were placed over the incisions. Skin sutures were removed about the fifth postoperative day. A total of 55 peripheral nerves were so severed and sutured in 10 animals.

The skin was tested at frequent intervals for areas of increased skin resistance after recovery of an animal from the anesthesia. When an area of high resistance was found, its borders were marked with a wax skin pencil. Record of the border was made by (1) drawing the zone on a standardized printed outline of the extremity, (2) writing descriptions in the protocols and (3) taking photographs of the extremity with the cutaneous marks present. In many specimens the borders were tattooed with india ink to facilitate the recognition of alterations in the patterns of skin resistance.

The instrument used in detecting the areas of altered resistance is a modification of the one described by Richter. Its essential parts are: a microammeter; four 4½ volt batteries; a 1,000 ohm potential divider, and electrodes. The modification consists in the addition of three 4½ volt batteries to the one 4½ volt

battery contained in the standard apparatus. A switch is provided so that 4½, 9, 13½ or 18 volts can be thrown into the circuit at will. A smooth increase of current of from 0 to 18 volts is obtained by the combined use of the switch and the potential divider. The amount of current flowing in the circuit at any given time depends on the resistance of the skin. The general rule followed in this study was that a sufficient potential to cause a current of 15 to 20 microamperes is placed in the circuit. Thus, gradations of increases in resistance can be detected by decrease in current through adjusting the current to give nearly full scale deflection of the microammeter needle in areas of low resistance. When searching a region suspected of having an elevated resistance, the corresponding normal area is tested first and the instrument adjusted so that the needle of the microammeter is deflected 15 to 20 microamperes each time the skin is touched with the movable electrode. The denervated area is then explored with the movable electrode. If no current flows, the area is described as having a high resistance; if some current flows, but the amount is clearly less than the corresponding normal area, the region is considered to have an intermediate resistance.

The principal technical problem encountered in making the skin resistance observations on monkeys is the production of adequate sweating. The animal must be sweating in order to lower the resistance of normally innervated skin sufficiently to permit detection of the high resistance of the denervated regions. Two methods of achieving sweating were tried: (1) application of external heat and (2) administration of "furmethide" (furfuryltrimethylammonium iodine), a parasympathetic-omimetic drug. The application of external heat was carried out by placing the animal under a surgical heat cradle containing a total bulb wattage of 440. Adequate sweating was detected by testing frequently the resistance of normally innervated skin until satisfactory lowering occurred. The application of heat was continued throughout the examination because a fall in body temperature produces an immediate cessation of sweating even with the internal body temperature higher than normal. Excessive application of heat was avoided also by reducing the amount in order to prevent direct stimulation of denervated sweat glands by a rise in local temperature.

Subcutaneous administration of 0.5 to 1 mg. of "furmethide" produces a profound sweating response in rhesus monkeys which reaches a maximum within five minutes or less. The amount of sweating so produced is adequate for studies of skin resistance. There is a disadvantage in the use of "furmethide," however, which caused its abandonment in favor of external heat in this study. Comparison of the borders of areas of high resistance obtained first after heating and then after administration of "furmethide" shows that for the first six to eight weeks after nerve transection the use of "furmethide" lowers the resistance of the denervated area to such an extent that differences from adjacent normal areas are difficult to detect. Prolonged application of external heat produces a similar lowering of resistance in denervated areas, but the effect is less pronounced and can be controlled by reducing the amount of heat. After six to eight weeks after operation, borders obtained by external heat and by furmethide are usually identical, but minor differences occur with sufficient frequency in rhesus monkeys to raise doubt concerning the accuracy of the skin resistance patterns obtained with the use of furmethide.

Dissections of the cutaneous branches of nerves to the extremities were made on material preserved in solution of formaldehyde U.S.P. The branches of the cutaneous nerves were followed as far as possible with the unaided eye to determine the general cutaneous area supplied by each peripheral nerve. Three to five dissections were made of each nerve. Composite drawings were then made of these

dissections to show the most common location and arrangement of the larger cutaneous branches.

The authors found that disturbances in sweat secretion in rhesus monkeys following transection of a peripheral nerve cause an elevation of the resistance of the skin to the passage of an electric current. The borders of such an area of altered conductivity show a general correspondence to the region of cutaneous supply of the severed nerve.

The elevation of resistance varies from that sufficiently high to cause almost no flow of current in the circuit to a state in which the amount of flow is only slightly less than in normally innervated skin. The areas of high skin resistance, determined by marking the site of first flow of direct current through the circuit, generally correspond to the anatomic area of autonomous supply described by others using different technics. Functionally, this is the area of anesthesia and anhydrosis. Areas of intermediate resistance, which surround an area of high resistance, correspond to the mixed, or intermediate, zone of a peripheral nerve. This zone represents anatomically the portion of skin supplied by both the transected nerve and the intact nerves adjacent to it; functionally, it is the region of hypesthesia and hypohydrosis. Intermediate areas of resistance have no sharp borders, as is the case in regions of high resistance.

The borders of areas of elevated skin resistance are static for only short periods. Immediately following section of a peripheral nerve, the area of high resistance is maximal. Soon, often twenty-four hours or less after operation, there may be detected a lowering of resistance at the periphery of the area of high resistance. This initial lowering is seen most prominently after section of a nerve supplying the palm or sole and is interpreted as being due to neural overlapping. The area of high resistance which remains after this initial lowering of resistance about its periphery thus represents the autonomous zone of the blocked nerve. In many nerves, particularly those supplying more distal portions of the extremities, a further lowering of resistance about the periphery of the area of high resistance becomes prominent fourteen to twenty-one days after operation. The possibility that this lowering may be due to ingrowth of sudorific fibers from adjacent intact nerves is suggested by the chronology and the symmetrically peripheral location of the change.

A slow shrinkage of the borders of the remaining pattern of high resistance occurs over a period of three to six months, depending on the nerve and the level of transection and suture. This process results in the disappearance of the region of increased skin resistance. This final slow shrinkage is interpreted as a functional manifestation of regeneration of the sudorific nerve fibers through the sectioned nerve.

GUTTMAN, Philadelphia.

THE EFFECT OF STILBESTROL ON RESISTANCE OF EXPERIMENTAL POLIOMYELITIS AND ENDOCRINOLOGY. F. J. CURLEY AND W. L. AYCOCK, ENDOCRINOLOGY **39**:414, 1946.

Aycock, in 1940, reported increased resistance of castrate monkeys treated with intramuscular injections of estrogen to intranasal inoculation of poliomyelitis virus. Curley and Aycock now report a series of experiments designed to emphasize the precise set of conditions under which this phenomenon occurs.

Immature normal male and female monkeys were treated with intramuscular injections of stilbestrol and testosterone proprionate, alone and in combination. Beginning on the tenth day of hormone treatment, all the animals were subjected

to repeated intranasal instillations of an emulsion of monkey poliomyelitis spinal cord. All the monkeys treated with stilbestrol alone became paralyzed; in 4 of 5 monkeys treated with testosterone alone and with stilbestrol and testosterone in combination poliomyelitis developed, and 3 of 5 monkeys not treated with the substances had the disease.

Male and female immature monkeys castrated two weeks before beginning glandular therapy were subjected to the same procedure. Of the stilbestrol-treated group, in 2 of 5 monkeys poliomyelitis developed; all the monkeys in the testosterone-treated group became ill; 1 animal in the stilbestrol-testosterone-treated group succumbed; and 4 of 5 monkeys in the control group became paralyzed.

The stilbestrol-treated monkeys which had resisted the intranasal instillations were inoculated intracerebrally with the emulsion. In all these the disease developed.

A group of 6 castrate female monkeys received intranasal instillations of the emulsion after a period of preliminary treatment with stilbestrol. After 19 instillations of virus during a fifty day period, 5 of 6 untreated control monkeys had poliomyelitis, while 2 of 6 stilbestrol-treated monkeys had the disease. The remaining monkeys were inoculated intracerebrally with the virus emulsion, and in all the animals experimental poliomyelitis developed.

The authors conclude that stilbestrol, alone or in combination with testosterone, produces an increased resistance to intranasal instillation of poliomyelitis virus in castrate monkeys. Testosterone alone has no protective effect. Neither substance increases resistance in the normal monkey. Repeated intranasal instillation of the virus does not serve to immunize, since later intracerebral inoculation resulted in experimental poliomyelitis in all the animals so treated.

FRANKEL, Philadelphia.

SOME OBSERVATIONS CONCERNING SPHINGOMYELIN AND SPHINGOMYELIN REINECKATE. M. H. HACK, J. Biol. Chem. **166**:455, 1946.

Sphingomyelin and sphingomyelin reineckates have been prepared from several sources by standard procedures and subjected to chemical analysis. Kjeldahl nitrogen values were found lower than Dumas nitrogen values, presumably owing to the presence of choline. Assay of sphingomyelin for choline yielded low values, presumably due to the resistance of phosphorylcholine to hydrolysis. Reineckates prepared from crude extracts contained both glycerin and hexose, implying, therefore, the presence of lipids other than sphingomyelin. The fraction of cephalin soluble in methyl alcohol and the cerebrosides kerasin and phrenosin formed reineckates. Recovery of pure sphingomyelins as the reineckate was low. The behavior of a saturated solution of ammonium reineckate in the presence of hydrochloric acid at 0 C. and the solubility of sphingomyelin in acid alcohol suggest the formation of an absorption complex, which was confirmed by leaching.

PAGE, Cleveland.

A MICROMETHOD FOR THE QUANTITATIVE PARTITION OF PHOSPHOLIPIDE MIXTURES INTO MONOAMINOPHOSPHATIDES AND SPHINGOMYELIN. G. SCHMIDT, J. BENOTTI, B. HERSHMAN AND S. J. THANNHAUSER, J. Biol. Chem. **166**:505, 1946

Thudichum long ago reported that sphingomyelin is much more resistant toward alkali than are the monophosphatides. Similar observations were made during a study involving the isolation of pure sphingomyelin from various organs. The difference between sphingomyelin and monophosphatides in regard to their behavior toward alkali was found to be so striking that the authors were encour-

aged to search for conditions permitting the application of this difference as a basis for the quantitative partition of phosphatide mixtures. It was found that lecithin and cephalin are quantitatively transformed into acid-soluble phosphorus compounds by normal potassium hydroxide at 37 C. within twenty-four hours. Under the same conditions, normal sphingomyelin forms no acid-soluble phosphorus compounds. A method for the quantitative microdetermination of sphingomyelin in lipid mixtures based on the selective saponification of the monophosphatides has been described. It was found that the lipids of the sciatic nerve of the cat and the rat contain a much higher percentage of sphingomyelin than those of the brain. The lipid fraction of the white matter of beef brain contains a much higher percentage of sphingomyelin than that of the cortex.

PAGE, Cleveland.

THE PENETRATION OF PENICILLIN THROUGH NORMAL AND INFLAMED MENINGES. J. MURRAY KINSMAN AND C. ANTHONY D'ALONZO, New England J. Med. **234**:459 (April 4) 1946.

The remarkable antibacterial effect of penicillin against many types of pathogenic organisms has been proved. However, there have been conflicting reports as regards the penicillin titer in the cerebrospinal fluid. Evidence has been presented which indicates that only extrathecal administration of penicillin is sometimes inadequate for the treatment of certain patients with meningitis. It is because of these conflicting reports that this study was undertaken.

In one series of experiments, more than 20 patients with early syphilis without meningitis were given intramuscular injections of 20,000 to 40,000 units of penicillin every three hours for one ot eight days, and the spinal fluid was assayed at various intervals from the fifth to the eighth day. In no case was penicillin detected in the spinal fluid.

In another series of experiments, specimens of the spinal fluid of 6 patients with meningococcemia who were being treated with penicillin were collected for assay. Five of the patients received an initial dose of 25,000 units intravenously, which was repeated one hour later and then every three hours day and night. The spinal fluid was collected at the eighth and the twenty-fourth hour of treatment in 4 cases and at the fifth and the ninth day in 1 case. The sixth patient was on the same schedule, but in this case each dose was 40,000 units and the spinal fluid was collected at the eighth and the twenty-fourth hour. In not 1 of the 12 specimens of spinal fluid was penicillin detected, although in every case the clinical response to treatment was excellent and in no case did meningitis develop.

Two patients with tuberculous meningitis were treated with large doses of penicillin given intrathecally; in spite of this, only a trace of penicillin could be found in the spinal fluid after eleven hours of treatment in 1 case and after twenty-three hours of treatment in the other.

The cases of 2 patients who had meningococcic meningitis were reported, and the data indicate that the penicillin levels and the cerebrospinal fluid were extremely variable. The authors conclude that penicillin penetrates the meningeal barrier too erratically to justify its use alone in purulent meningitis.

Four main points indicating that extrathecal therapy alone is not effective in meningitis have emerged from the present studies and from previous reports. These are the development of pneumococcic meningitis in patients receiving penicillin extrathecally for pneumococcic pneumonia; the failure of many patients with pneumococcic and meningococcic meningitis to respond to penicillin until it was administered intrathecally; the low concentrations and irregularity of appearance

of penicillin in the spinal fluid of patients with meningitis following extrathecal administration alone, and the fact that many cases of meningitis are caused by meningococci that are relatively resistant to penicillin.

It is the opinion of the authors that patients with pneumococcic, streptococcic or staphylococcic meningitis should receive penicillin by both the extrathecal and the intrathecal route. For meningococcic meningitis, sulfadiazine is probably preferable to penicillin, but here, too, if penicillin is administered, it should be given both extrathecally and intrathecally.

GUTTMAN, Philadelphia.

THE EFFECT OF AGE, HEAD RESISTANCE, AND OTHER PHYSICAL FACTORS ON THE STIMULUS THRESHOLD OF ELECTRICALLY INDUCED CONVULSIONS. D. WATTERSON, J. Neurol., Neurosurg. & Psychiat. **9**:121 (Jan.) 1946.

Using a constant stimulating voltage, Watterson investigated the threshold duration of stimulus for induced convulsions in 70 psychotic male subjects. By standardizing the technic, the author attempted to eliminiate all variables other than the physical variables of the patient himself. These included age, head resistance, height, weight, interelectrode distance. He found that the convulsion threshold was not directly correlated with height, weight or interelectrode distance. There was, however, a definite correlation between threshold and head resistance and between threshold and age, but not between age and head resistance. The correlation between age and convulsion threshold appeared to be linear and seemed to depend on changes in the brain, rather than on superficial tissues of the head. This was significant as suggesting a possible relation between the slowly decreasing sensitivity of the central nervous system to electrical insult and known changes in psychologic behavior with age.

N. MALAMUD, San Francisco.

Neuropathology

DISSEMINATED, DIFFUSE AND TRANSITIONAL DEMYELINATION OF THE CENTRAL NERVOUS SYSTEM. L. ROIZIN, M. HELFAND and J. MOORE, J. Nerv. & Ment. Dis. **104**:1 (July) 1946.

Roizin, Helfand and Moore summarize the extensive literature on the demyelinating encephalopathies. They point out that the wealth of published material has served rather to confuse than to clarify the various controversial aspects. As a result, similar clinicopathologic entities have been given different names. These include diffuse cerebral sclerosis, encephalitis periaxialis diffusa, encephalitis periaxialis scleroticans, symmetric cerebral centrolobar sclerosis, encephalomyelomalacia chronic diffusa, encephaloleukopathia scleroticans progressiva, sclerotic inflammation of the white matter of the hemispheres, progressive degenerative subcortical encephalopathy, encephalopathia subcorticalis diffusa, encephalitis periaxialis concentrica, leukoencephalopathia myeloclastica primitiva, acute leukoencephalopathia, encephalopathia periaxialis diffusa, diffuse infiltrative encephalomyelitis, familial diffuse sclerosis of the brain, infantile type, aphasia axialis extracorticalis congenita, familial diffuse sclerosis of the brain, juvenile type, leukodystrophia cerebri progressiva hereditaria, and familial progressive leukodystrophy. Ferraro has attempted to bring some order into this confusion by a classification dividing the demyelinating diseases into sporadic and familial types. Each of these is subdivided into types with patchy and diffuse distribution of disease, and these subvarieties are further divided into acute, subacute and chronic varieties. Putnam has claimed

that the one factor common to all the demyelinating diseases is their vascular origin in the sense that venous thrombi, especially in the early stages, can always be detected histopathologically.

Four cases are reported, each with elaborate histopathologic studies. Of these, 2 were considered to represent multiple and transitional sclerosis, and 2 diffuse sclerosis. The authors agree with the opinion of many workers that there is a close relation between disseminated and diffuse demyelination as far as hereditary traits, sex, age, duration of disease, clinical features and gross pathologic changes are concerned.

Histopathologically, the demyelinated areas may be differentiated as follows: (*a*) Multiple or patchy demyelination, variable in form and size. At times neighboring small areas coalesce, forming larger ones, which gradually may involve very large areas of the white matter, forming the so-called transitional type. (*b*) Diffuse demyelination, which may involve the whole subcortical white matter with the exception of the U fibers. This type includes Schilder's disease. (*c*) Diffuse demyelination, with interspersed islands of more or less preserved myelin sheaths generally surrounding blood vessels (Pelizaeus-Merzbacher variety. (*d*) Concentric demyelination formed by bands of more or less normal myelin alternating with demyelinated stripes (Baló or Marburg type).

CHODOFF. Washington, D. C.

CUSHING SYNDROME AND HYALINIZATION OF THE BASOPHILIC CELLS. M. BARRETTO NETTO, Arch. brasil. de med. **34**:169 (May-June) 1944.

A mentally retarded man aged 30 was admitted to the hospital with a history of convulsive seizures. Anamnestic studies were impossible because of his mental state. He weighed 74 Kg. and was 163 cm. in height. Examination revealed an abundance of fat in the subcutaneous tissues and hypertrichosis. Lumbar pucture yielded fluid with a lymphocyte count of 0.9 per cent; the Wassermann reactions of the spinal fluid and the blood were negative. Autopsy revealed chronic leptomeningitis and atrophy of the frontoparietal convolutions. Fatty liver, lipid nephrosis, pulmonary congestion, hypertrophy of the heart and subacute splenitis were also noted. Microscopic examination of the hypophysis showed a basophilic adenoma. Eosinophilic cells were absent and altered basophilic cells abundant. The author notes the nuclear changes in the hyalinized cells, previously reported by Severinghaus (Cytology of the Pituary Gland, *A Research Nerv. & Ment. Dis., Proc.* [1936] **17**:69, 1938). In the absence of a definite Cushing syndrome, this case is similar to the one reported by Ecker (*Endocrinology* **23**:209 [Nov.] 1938). In the region of the adenomatous nodule, thickening of the blood vessels was noted; there were no red cells in any of these vessels, in contrast with the vessels which were not near the adenomatous nodule. The author believes that the hyaline changes are due to ischemia resulting from alterations in blood vessels.

N. SAVITSKY, New York.

THROMBOANGIITIS OBLITERANS OF THE BRAIN. F. LLAVERO, Schweiz. Arch. f. Neurol. u. Psychiat. **57**:290, 1946.

Llavero regards thromboangiitis obliterans as a reaction with many possible causes. Among the exogenous agents, the importance of which he thinks has been generally overrated, he mentions nicotine, infection and exposure to cold. After discussing the question of allergy at some length, he expresses the conviction that angiospasm plays an important role in initiating the characteristic anatomic changes in the intimal layer of the affected vessels.

Two cases are reported. In the first case, that of a man aged 44, right hemiplegia and aphasia suddenly developed four hours after he received an injury to his head which did not render him unconscious. He recovered within four days and was free from symptoms until three years later, when he began to complain of frontal headache, vertigo and paresthesias in the right leg. The right lower limb became progressively weaker, and the patient again became aphasic. Examination revealed emotional incontinence, slowing of the mental processes, parkinsonian tremor of both arms and spastic tetraparesis, with pronounced spastic weakness of the right lower limb. As the needle was withdrawn after an unsuccessful lumbar puncture with the patient in the sitting position, he collapsed. He recovered promptly, but ten minutes later passed into a state of deep coma, with stertorous breathing, rolling movements of the head and tonic spasms, which were pronounced in the upper extremities. He died the following day. In addition to changes characteristic of thromboangiitis obliterans in the aorta and the femoral and subclavian arteries, postmortem study revealed the presence of organized and recanalized thrombi in the internal carotid arteries. The obliterating process in the intracranial arteries was limited to the large vessels of the base of the brain. Edema of the brain, although generalized, was more marked in the right hemisphere. Multiple foci of softening, which involved mainly the corpus striatum and adjacent white matter, on the other hand, were considerably more extensive on the left side. In addition, typical plaques of multiple sclerosis were observed about the posterior horns of the lateral ventricles and also in the spinal cord. Miscroscopic studies of small vessels in and about the plaques were evidently not undertaken.

In the second case, that of a man aged 58, an apoplectic seizure was followed by paralysis of the right arm and severe aphasia. A year later the patient was readmitted to the hospital because of mental confusion and died within a few days. Examination of the brain revealed multiple areas of softening, both old and recent, and granular atrophy of the cerebral cortex in regions where the three cerebral arteries enter into anastomotic relation. Changes considered characteristic of thromboangiitis obliterans were limited to the cerebral arteries, but, in contrast to observations in the first case, the cortical vessels, as well as those of the base, were diffusely involved.

The author comments on the discrepancy frequently observed between anatomic changes and clinical signs in cases of thrombangiitis obliterans of the brain. He also emphasizes the variable character of neurologic and psychiatric symptoms.

DANIELS, Denver.

Psychiatry and Psychopathology

THE DISORGANIZATION OF BEHAVIOUR IN FATIGUE. D. R. DAVIS, J. Neurol., Neurosurg. & Psychiat. **9**:23 (Jan.) 1946.

Davis tested 34 R.A.F. pilots with the Cambridge cockpit to determine the effect of fatigue on the organization of a relatively complex skill, namely, instrument flying. It was noted that skilled activity became disorganized and showed two phases in this disorganization (1) a phase of "overactivity," characterized by increases in the number of control movements and corrections, and (2) a phase of "withdrawal," characterized by a decrease in the number of control movements and corrections.

Based on these reactions, a classification of 268 fliers was made into normal, overactivity and withdrawal groups. In the overactivity group all parts of the work were similarly affected; errors became more frequent and were overcorrected;

activity became excessive and movements of the controls tended to be too extensive, associated with an increase in restless and inappropriate movements. In this group, there was emotional tension and anxiety, along with increased muscle tone, flushing of the face and frequent sweating. In the withdrawal group some parts of the work were more affected than others; errors were less frequent but their correction was tardy; movements of the controls were excessive but fewer. This was associated with a lessened interest, and emotional reaction and flushing or sweating were rarely seen. The observations suggested that the effects were of the same kind as those generally occurring in states of fatigue, whether this is applied to physiologic exhaustion or to psychologic frustration. The emotional disturbance was the most important feature of the overactivity reaction, resulting, apparently from heightened expectancy, while the withdrawal reaction represented psychologically a state of reduced expectancy.

N. MALAMUD, San Francisco.

PSYCHOTHERAPY OF ULCERATIVE COLITIS. RANYARD WEST, Lancet **2**:899 (Dec. 21) 1946.

West discusses 3 cases of ulcerative colitis from the point of view of brief psychotherapy. He states that therapy, although it should be based on knowledge of the freudian unconscious, can be successful if it gives acceptable interpretations to the patient of his symptoms and gives him crucial reassurances. He adds that too early or too deep psychoanalysis may expose more unconscious problems than it can alleviate and thus may aggravate, even dangerously, the course of the physical illness.

The first patient obtained relief after two sessions of psychotherapy. The first session, after the history was taken, consisted of an introductory explanation of the effects of emotion on bodily states, including the eliminative processes. The second interview contained a rough classification of character types and a further account of the functions of the colon and its response to emotion. Immediately after this second interview, the stools became normal in freqeuncy, signs of anxiety vanished and the patient ate well and gained weight. The other 2 patients received about twenty hours of similar therapy and both responded well, but 1 passed into a psychotic state with marked guilt feelings about her treatment of her father and became demented and bedridden.

A brief history of the development of recognition of emotinal disturbances in ulcerative colitis is given. The mechanism of the disease is discussed, and the animal experiments of Luim and Porter, in 1939, are cited. They made explants of the dog's colon and noted that ulceration of the mucosa could develop within twenty-four hours of the operation if the colonic muscle remained in spasm. The ulcers could heal in from ten to fourteen days if the muscle became relaxed. The author states that it does not seem an improbable explanation of some cases of ulcerative colitis that anxiety causes muscular spasm and mucosal changes in an organ previously rendered susceptible by either a psychologic or a physical sensitization, or by both.

MADOW, Philadelphia.

GRIESINGER AS PRECURSOR OF FREUD. PAULO LENTINO, Arq. neuro-psiquiat., Sao Paulo **4**:421 (Dec.) 1946.

Lentino found many statements in Griesinger's "Pathologie und Therapie der psychischen Krankheiten" to indicate that he was aware of certain psychologic facts which were later included in Freud's teachings. The author used the French

translation of this German textbook, which was published in 1845. On page 30 of the French translation is a perfect definition of the dynamic unconscious and its significance in mental life. On page 38, the problem of repression of instincts is clearly stated. On page 44, hunger and love as motivations of conduct are discussed. On page 45, he refers to the mechanism of repression and even uses the term "complex," which was later coined by Jung. On page 47, he discusses catharsis and sublimation in a manner similar to Freud's. On page 49, he indicates the importance of a well integrated, strong ego. On page 56, he points out that anxiety and depression can result from conflicts. On page 60, he states that mental disease may result from failure to realize ambitions. He also discusses dreams as wish fulfilment. The author does not believe Freud read Griesinger directly. He thinks some of the ideas of Griesinger were due to the influence of Schopenhauer.

N. Savitsky, New York.

Cholesterol in Spinal Fluid in Some Psychoses. Gustavo Mujica Cervantes, Rev. de psiquiat. **10**:97, 1045.

For 4 normal controls the cholesterol of the spinal fluid was 0.30 mg. per hundred cubic centimeters. Fifty-two psychotic patients were studied. In 19 schizophrenic patients there was a definite increase in the amount of cholesterol, with higher values associated with more recent onset. For 5 patients with duration of the illness of from one to twelve months, the average cholesterol reading was 3.66 mg.; for 6 with durations of two to six years, the average was 2.48 mg., and for 8 with durations from ten to twenty-nine years the average was 2.25 mg. per hundred cubic centimeters. For 5 patients with so-called chronic delirium (paraphrenia and paranoia) the average was 2.52 mg. per hundred cubic centimeters. The average for 8 epileptic patients with psychoses was 4.44 mg.; for 5 patients with senile psychosis with cerebral arteriosclerosis, 3.28 mg.; and for 1 patient with senile psychosis of eighteen months' duration, 5.16 mg. per hundred cubic centimeters. For 5 syphilitic patients the values were 2.88 mg., and for 7 patients with alcoholic psychoses, 2.91 mg. per hundred cubic centimeters. One patient with traumatic psychosis showed 3.96 mg.; 1 with psychopathic personality, 2.04 mg., and 1 with reactive depression, 0.30 mg. per hundred cubic centimeters. The cholesterol value in the spinal fluid in psychoses is, therefore, three to thirty times normal. This increase seems to parallel the activity of the psychosis.

N. Savitsky, New York.

Meninges and Blood Vessels

Meningococcic Infections with Special Reference to Certain Diagnostic Considerations: An Analysis of Thirty-Seven Cases. Albert C. Oxman and Isidore Fischer, Ann. Int. Med. **23**:924 (Dec.) 1945.

The purpose of this communication is twofold: to emphasize the clinical syndrome of meningococcic infection, particularly of the sporadic and the nonmeningitic variety, and to illustrate by case reports the various atypical forms which this infection may assume. Additional data on the effectiveness of chemotherapy in this disease are presented incidentally.

Thirty-seven patients with meningococcic infection, in 9 of whom meningeal localization did not occur, were observed at the Station Hospital, Camp Bowie, Texas, during the thirteen month period ending Jan. 24, 1944. Four of

the 9 patients with meningococcemia, of whom 1 died, and 23 of the 28 patients with meningitis, of whom 1 died, were treated during the period of national epidemic incidence; the other cases occurred sporadically.

Two of the patients had an overwhelming type of infection and presented the so-called Waterhouse-Friderichsen syndrome. In 1 patient the acute meningococcemia was unrecognized and, therefore, was inadquately treated. The subsequent circulatory collapse was probably due to adrenal failure, which is not uncommon in severe infections. This patient recovered after therapy with intravenously injected sufadiazine. Blood plasma, oxygen and epinephrine were necessary.

In another case there was an acute, uncomplicated meningococcemia which, within seven hours, passed into meningitis. One patient was confused and excited, and the diagnosis of acute meningitis was established later. In another case the blood culture was sterile. Negative laboratory evidence cannot exclude the possibility of chronic meningococcemia. Other cases of meningococcemia with sterile blood cultures are reported.

The authors conclude that the successful therapy of meningococcemia depends on two considerations: Prompt and adequate chemotherapeutic measures, which imply the initial administration of the sulfonamide drugs by the parenteral route if necessary. The institution of chemotherapy, however, depends on early recognition of the disease, preferably before meningeal involvement occurs. Such recognition is achieved only by the constant recollection that meningococcic infection can mimic any acute infectious process, can present superficially a picture of mental illness and is frequently introduced by nasopharyngitis, headache and backache, all of which are symptoms of many other, much less serious, diseases.

GUTTMANN, Philadelphia.

RECURRENT MENINGOCOCCAL MENINGITIS. LOUIS WEINSTEIN and EDITH DUMOFF-STANLEY, New England J. Med. **234**:364 (March 14) 1946.

Weinstein and Dumoff-Stanley report a true recurrence of meningococcic meningitis in a woman aged 22 who was in a nephrotic stage of chronic diffuse glomerulonephritis. This patient had two attacks of meningococcic meningitis during a period of seven months. The source of the bacteria in the two episodes of the disease is unknown. It is deemed likely that a carrier was the source of infection, and there is some bacteriologic evidence of a chonic carrier state in the patient. During the course of the illness three episodes of severe bacterial infection exerted no noticeable effect in the course of the glomerulonephritis.

The question of the relation of the nephrotic stage of the chronic diffuse glomerulonephritis to the increased susceptibility to bacterial infection is interesting, but no concrete evidence for a causal connection between the two is at hand. Whether an analogy can be drawn between lipid nephrosis and the nephrotic state of chronic glomerulonephritis with respect to decreased resistance to infection is difficult to say.

GUTTMAN, Philadelphia.

ACUTE INFECTIOUS LYMPHOCYTOSIS IN ENGLAND. ALEX J. STEIGMAN, Lancet **2**:944 (Dec. 28) 1946.

Steigman states that acute infectious lymphocytosis was first described in children by C. H. Smith in the United States in 1941. The clinical signs and symptoms are few, and include mild symptoms of the upper respiratory tract, low grade fever, cervical adenopathy, irritability, abdominal pain and meningeal irri-

tation. The diagnosis is established on routine laboratory examinations which show 12,000 to 100,000 leukocytes with 50 to 90 per cent small mature lymphocytes. There is absence of anemia, thrombopenia or heterophil antibody agglutination. These findings may last several months.

The disease appears to be communicable, because it has occurred in institutional outbreaks. The incubation period is from twelve to twenty-one days. The mode of transmission is unknown, and treatment is symptomatic. Lymph node biopsies, done by Smith in 1944, showed degeneration of lymph follicles with proliferation of the reticuloendothelium of the sinuses. No report of neurologic or spinal findings was given.

MADOW, Philadelphia.

COCCIDIOIDAL BRAIN ABSCESS. A. E. RHODEN, Bull. Los Angeles Neurol. Soc. **11**:80 (March-June) 1946.

Rhoden describes what he regards as the first case of coccidioidal abscess of the brain to be reported in the literature. The abscess had almost replaced the tissues of the left cerebellar hemisphere and caused compression of the fourth ventricle and terminal meningitis. The clinical course was insidious and asymptomatic up to the final stage, when mounting intracranial pressure and meningitis became manifest. The lesion occurred in a 16 year old white boy who had lived in the endemic area for about three years and had severe diabetes mellitus.

J. A. M. A.

CEREBRAL VASCULAR OCCLUSIONS IN YOUNG ADULTS. IRA S. ROSS, J. Nerv. & Ment. Dis. **104**:51 (July) 1946.

Ross reports the occurrence of destructive vascular lesions in young adults without subarachnoid bleeding and without any of the usual causes, such as syphilis, hypertension, bacterial endocarditis or blood dyscrasia. In all cases, on clinical grounds, the vessel involved appeared to be of comparable size. It appeared unlikely that either vascular spasm or congenital aneurysm was responsible, and the author believes that the responsible lesion was arterial occlusion secondary to a patch of localized atherosclerosis.

CHODOFF, Washington, D. C.

SOME OBSERVATIONS ON THE VISUAL FIELDS IN HYDROCEPHALUS. E. B. C. HUGHES, J. Neurol., Neurosurg. & Psychiat. **9**:30 (Jan.) 1946.

Using methods of quantitive perimetry, Hughes investigated defects of the visual field which occur in association with hydrocephalus and distention of the third ventricle resulting from intracranial tumor. He excluded from the study cases of secondary optic atrophy. The kinds of change in the visual fields could be distinguished: (1) scotomatous type, characterized by a localized amblyopic zone, either relative or absolute, which affects all isopters of all visual angles normally seen in the area, and (2) depression type, in which all isopters over a wide area of the field are affected simultaneously by an indrawing of the isopters and by "graying" of the whole field. A progressive change takes place, in which the earliest manifestation is a very small paracentral scotoma at the apex of the lower temporal quadrant. This is followed by a depression type defect, at first in the lower and later in the upper temporal quadrant, but at no time resulting in a complete temporal hemianopsia. The type of field defect is dependent on the site of the lesion. If the chiasm is prefixed, a scotomatous change will occur, and if

postfixed a depression type of field defect will result. In secondary atrophy of the optic nerve binasal field defects are usually observed and in this way can be distinguished from the changes in simple hydrocephalus.

N. Malamud, San Francisco.

Diabetes Insipidus in Association with Postpartum Pituitary Necrosis. A. W. Spain and F. Geoghegan, J. Obst. & Gynaec. Brit. Emp. **53**:223 (June) 1946.

According to Spain and Geoghegan, it has been established that postpartum ischemic necrosis of the anterior lobe of the pituitary gland is a relatively frequent complication in cases in which delivery is attended with a severe degree of collapse. The striking clinical feature of the 2 cases reported by them was the occurrence of an extreme degree of polyuria and the observation at necrospy of necrosis in both the anterior and the posterior lobe of the pituitary body. The occurrence of symptoms of diabetes insipidus in association with postpartum necrosis of the pituitary has not previously been commented on. Profound shock occurred at the time of delivery in both cases. In the first case this was associated with acute inversion of the uterus and in the second case with combined accidental hemorrhage occurring in unusually unfavorable circumstances.

J. A. M. A.

Anatomicoclinical Observations in a Case of Parinaud Syndrome: Role of the Region of the Posterior Commissure. A. Devic, Paufique, P. Girard and P. Guinet, Rev. neurol. **77**:37 (Jan.-Feb.) 1945.

A man aged 37 had been shot in the head at the age of 18. Four years before his admission right hemiplegia and left squint appeared. One month prior to admission he had headache, vomited and became comatose. Examination on his admission showed right hemiparesis and complete paralysis of the left third nerve with external strabismus. There were paralyses of all ocular muscles except the left external rectus. The left pupil was dilated and fixed to light and in accommodation. There was no disturbance in lateral conjugate gaze, but there was complete paralysis of upward and downward gaze. Voluntary and reflex vertical conjugate movements were impaired. The corneal reflexes were present, and the fundi were normal. The visual fields were intact. Roentgenograms showed the bullet to be in the midline, a little behind and above the sella turcica. Ventriculograms showed that the ventricles were distended but not deformed. The bullet was in the region of the posterosuperior part of the third ventricle. The patient died after the ventriculographic examination.

Autopsy showed the bullet to be in the posterior part of the third ventricle, in contact with the posterior commissure, but not cutting it. There were small areas of softening in the left peduncle and in the left wall of the third ventricle. The area of softening in the peduncle destroyed the nucleus of the third nerve inferiorly. The posterior longitudinal bundle seemed only partially involved. The focus extended in a narrow strip rostrally to the wall of the third ventricle, near the ependymal lining. The posterior commissure and the quadrigeminal tubercles were not involved by the focus. The lesion was juxtacommissural. Degenerative changes were observed in the right oculomotor nucleus and in the rostral two thirds of the posterior commissure. There were no degenerative changes in the posterior longitudinal bundle. This case illustrates the role of the posterior commissure in paralysis of vertical gaze. It is impossible to state from the available facts whether the nuclei or the connecting fibers played the dominant role.

N. Savitsky, New York.

RELATIVE HEMIANOPSIA. F. THIEBAUT and L. GUILLAUMAT, Rev. neurol. **77**:129 (May-June) **1945.**

Relative hemianopsia is defined as the type of hemianopic field defect which is discovered during simultaneous exploration of both fields of one eye or both temporal or both nasal fields of both eyes. Such relative field defects may be missed during ordinary perimetric examination of one field at a time. Relative hemianopsia is occasionally of localizing value. The authors cite 2 cases illustrating the usefulness of relative hemianopsia in diagnosis. In 1 case there were signs on the left side referable to the pyramidal tract and increased intracranial pressure. The observation of a right relative hemianopsia led to a decision to make a ventriculographic examination, which demonstrated a tumor in the left temporal region. The other case was that of a child with a syndrome of increased intracranial pressure but with no localizing signs. The ventriculogram was difficult to interpret. The discovery of right relative hemianopsia pointed to a lesion on the left side. At operation a tumor in the left frontal region was removed. While the phenomenon of relative hemianopsia has long been known, it has not been sufficiently emphasized in clinical practice.

N. SAVITSKY, New York.

SYNDROME OF TEMPORAL ACCELERATION AND RETARDATION OF TIME. HANS HOFF and ABRAHAM MOSOVICH. Rev. argent. de neurol. y psiquiat. **10**:162 (Sept.-Dec.) 1945.

On coming out of an insulin coma, a patient repeatedly complained that objects around him were moving slowly and people rapidly. This was accompanied with some micropsia and macropsia. The neurologic examination showed hemianopsia and signs referable to the pyramidal tract on the left.

A 25 year old merchant began to feel ill while traveling by plane two days after his marriage. Gastrointestinal distress, general malaise and chills were present. While driving his car from the airport, he felt he was traveling very fast, though he was actually slowing up. Objects to the right and left of him seemed to be moving very fast, while those directly ahead seemed stationary. On the way he had to cross a familiar bridge, which now seemed too narrow. On reaching home, his temperature was 100 F.; there were general malaise, chilliness and pain. Two hours later the pain became worse; diarrhea appeared, and he complained of dizziness. He was operated on for appendicitis the next day. After the operation, he felt very weak and had difficulty in standing and walking, especially with the eyes closed; he complained of tremulousness and almost constant somnolence. There were some urinary incontinence and dysuria from the onset. Three months later intense thirst developed (5 liters a day), and he began to gain weight. There was gradual improvement.

A 62 year old man stated that ten years previously, while seated in a cafe, he became dizzy. On emerging into the street, he thought the cars to the right and left of him were moving very rapidly; objects directly ahead of him seemed to move at a normal pace, or even slowly. The same was true of pedestrians to his right and left and in front of him. This experience cleared up in five days. A left hemianopsia was found at the time. Ten years later, after sudden occipital pain, he noted that some one coming toward him was moving very slowly and that another person on his right moved rapidly. On admission, he complained that the nurse and doctor were moving so fast they were making him dizzy. Outdoors, automobiles and pedestrians in the periphery of his fields seemed to move very fast, while objects in the center moved with funereal slowness. Examination

showed facial paralysis and hemianopsia on the left side. The day after admission left hemiplegia appeared. He died a few days later, with coronary thrombosis. Autopsy showed complete destruction of the third occipital convulution, extending to 1 cm. from the occipital pole and to the second temporal convolution anteriorly on the right side. The authors postulate a mechanism, analogous to a movie projector, which regulates velocity and frequency of movement. The center for this mechanism is in the region of the calcarine fissure. When the calcarine fissure itself is involved, hemianopsia is also present.

N. SAVITSKY, New York.

CHORDOMA OF THE SELLA TURCICA. RAFAEL J. BABBINI and ANIBAL CASTANE DECOUD, Rev. argent. de neurol. y psiquiat. **10**:169 (Sept.-Dec.) 1945.

The case of a man aged 45, with a history of symptoms of seventeen years' duration, is reported. During December 1927 he fell a distance of 9 meters, landing on his head. He was unconscious for ten minutes. There were apparently no sequelae. On April 2, 1929 he began to complain of paroxysmal frontal and occipital headaches with diplopia. The attacks recurred once or twice a year until 1937, when bilateral ptosis was noted during an attack. He continued to have an occasional attack of headache with diplopia until June 1944, when he noted diminution of vision in the left eye. During September 1944 impairment of vision in the right eye was observed. The last attack of headache with diplopia occurred during July 1944, followed by persistent squint. When he was seen on Nov. 28, 1944, there was paresis of the superior, inferior and lateral rectus muscles on the left side. The left superior oblique muscle was not involved. The left eye was blind, with atrophy of the disk and significant thinning of the arteries. The left pupil was in mydriasis and failed to react to light or in accommodation. The right pupil reacted sluggishly to light and accommodation; there was paralysis of the external rectus muscle and of the elevator muscle of the lid. The right disk was pale, and central vision was greatly impaired, with a pronounced temporal defect.

A roentgenogram of the skull during June 1937 showed that the anterior clinoid processes were intact; there was thinning of the posterior clinoid processes. The bone behind the sella contained a number of large cavities. The sphenoid sinus was separated from the sella by an area of osseous condensation. A roentgenogram of the skull on Nov. 21, 1944, showed destruction of the anterior clinoid processes; the posterior clinoid processes were blurred; the sphenoid sinus was reduced in size, and the floor of the sella was condensed. The cavities previously noted had disappeared.

An operation was performed on Dec. 2, 1944. A frotoparietotemporal flap was raised on the left side and the frontal lobe lifted, disclosing a grayish tumor which invaded the sella. The tumor raised the chiasm and flattened the left optic nerve. A large part of the tumor was removed. Microscopic study showed the tumor to be a chordoma. Postoperative roentgenotherapy was given.

N. SAVITSKY, New York.

OPHTHALMOLOGIC SYNDROMES ASSOCIATED WITH TUMORS OF THE FRONTAL LOBE. MAGIN A. DIEZ, Rev. Asoc. méd. argent. **60**:194 (April 15) 1946.

Diez reviews the ocular findings in 10 cases of verified tumor of the frontal lobe. In all cases papilledema was present bilaterally. Edema usually appeared first and was more pronounced on the side of the tumor. The Foster Kennedy syndrome was not observed in this series. In 4 cases visual acuity was normal, and

in 6 cases it was impaired, chiefly on the side of the tumor. The blindspots were enlarged in proportion to the extent of the papilledema. Central scotoma was found in 4 cases—in 2 on the same side as the tumor, in 1 on the opposite side and in 1 bilaterally. Bitemporal hemianopsia was found in 1 case, and atypical nasal hemianopsia, in 2 cases. There was some degree of concentric contraction of the fields, especially on the nasal side in 1 case. In 2 cases there was paresis of both external rectus muscles. In 1 case mild ptosis was noted on the side of the tumor, and in another, convergence insufficiency. The author also observed nystagmus on extreme lateral gaze, especially toward the side of the tumor. The pupillary findings were abnormal in 4 cases.

N. Savitsky, New York.

Diseases Of The Brain

Hemiballism. Hugo Lea-Plaza and Enrique Uiberall, Rev. de psiquiat **10**:75, 1945.

Greiff in 1883 first noted a relation between a lesion in the corpus luysi and hemiballism. Since then 50 cases have been reported with anatomic verification. The authors report 3 more cases, 2 of which were verified at autopsy. Right-sided hemiballism in a man of 75 cleared up in seven weeks. The second case was that of a man of 55 who died five days after the sudden appearance of hemiballistic movements. A day or two before the appearance of this dyskinetic syndrome, there was hemiparesis on the same side. At autopsy, there were multiple foci of softening throughout the brain and a recent hemorrhagic lesion in the region of corpus luysi spreading to the pyramidal tract at the base of the peduncle. The hemorrhage spread caudally to involve the upper third of the substantia nigra and rostrally to the fields of Forel and the zona incerta. The hemorrhagic focus displaced the mamillothalamic tract medially. The third case was that of a man aged 56 who died six days after the onset of the dyskinetic syndrome. The blood pressure was 220 mm. At autopsy there were advanced arteriosclerosis of the cerebral vessels and an old cystic vascular lesion in the putamen and globus pallidus. The subthalamic nucleus on one side was destroyed by a recent hemorrhage, which extended to the base of the peduncle, the dorsolateral part of the substantia nigra, a small part of the field of Forel and the red nucleus. The fact that the movements disappear with involvement of the pyramidal tract suggests a cortical origin. The authors note the similarity of the patterns of the hemiballistic movements to voluntary motion. They believe that the normal inhibitory role of the corpus luysi is removed when it is destroyed.

N. Savitsky, New York.

Diseases of the Spinal Cord

Medicolegal Aspects of Spinal Cord Injuries. Foster Kennedy and Peter G. Denker, J. Nerv. & Ment. Dis. **103**:667 (June) 1946.

Kennedy and Denker review briefly the structure and functions of the spinal cord as a preliminary to a critical examination of the relation of injury and various diseases of the spinal cord.

Aftr trauma to the spinal cord, symptoms must appear almost immediately to be properly attributed to the injury. Exceptions to this are Kummel's disease

and meningitis circumscripta serosa, in both of which manifestations of injury to the spinal cord may be late results of injury.

In multiple sclerosis, injury is much more likely to be due to preceding weakness than the cause of it. The authors, citing the failure of the disease to increase in frequency during World War I, feel that injury plays no role in the production of multiple sclerosis. There is also no relation between injury and the production of amyotrophic lateral sclerosis or acute anterior poliomyelitis. In rare cases injury may be responsible for aggravating the symptoms of tabes dorsalis, but can never be said to have a causal relation. Although syringomyelia is not caused by injury, sudden hemorrhage in a preexisting cavity of the spinal cord may occur with severe trauma. Injury plays no part in the causation of tumors of the spinal cord.

CHODOFF, Washington, D. C.

LATERAL COLUMN DEGENERATION FOLLOWING NUTRITIONAL DEFECTS WHILE PRISONERS OF WAR IN THE FAR EAST. G. RIDDOCH, Proc. Roy. Soc. Med. **39**:598 (Aug.) 1946.

Two patients, aged 33 and 27 years, who had lived on a diet consisting chiefly of polished rice, manifested symptoms of mental disturbance, visual errors, muscular weakness, urinary incontinence, cutaneous lesions, glossitis and sores of the mouth. They now show a pure lesion referable to the pyramidal tract in both lower extremities.

BERRY, Philadelphia,

TUBERCULOUS SPINE WITH PARAPLEGIA. V. C. SNELL, Proc. Roy. Soc. Med. **39**:599 (Aug.) 1946.

A woman, aged 21, in October 1941 complained of aching in her back. In February 1942 roentgen examination revealed a partial collapse of the seventh thoracic vertebra, and a week later paraplegia began to develop. A sensory level at the fifth and sixth thoracic segments, was complete by May 1942. In July 1943 a laminectomy was performed to relieve pressure on the cord; no pus was obtained. No improvement followed, and in December 1943 costotransversectomy on the right side revealed a large abscess cavity containing about 1 Gm. of semicaseous pus. This was evacuated. Slow recovery ensued; the spasticity disappeared in November 1944, and, with the aid of a double iron and toe-lifting strap on the left leg, she was discharged, walking, in March 1945.

BERRY, Philadelphia.

REFRACTORY SCIATICA DUE TO INTRASPINAL EXTRADURAL NEURINOMA. JULIO DIEZ, Rev. Asoc. méd. argent. **60**:171 (April 15) 1946.

Diez reports 2 cases of monosymptomatic unilateral refractory sciatica resembling the clinical picture of herniated disk. The first case was that of a 35 year old woman who complained of sciatic pain on the right side and paresthesias in the right toes for three years. During April 1942 the pain became worse and more constant; it was intensified with flexion of the trunk and extension of the right lower limb. There was occasional sudden weakness of the right lower limb. At times the patient was unable to straighten up from the bent position because of pain. The pain was also noted as worse on flexion and extension of the head. Examination showed spasm of the paravertebral muscles and positive Laségue and Naffziger signs. The right ankle jerk was diminished; there were atrophy of the right calf and weakness of the extensors of the right foot and mild diminution for

touch and pain sensation in the last three toes of the right foot. The spinal fluid was normal. A roentgenogram after a contrast medium was injected into the spinal canal revealed a small indentation on the oblique view at the level of the fifth lumbar vertebra, identical with that seen in herniated disk. An operation was performed, and a small extradural cystic neurinoma, 16 by 12 mm., was seen attached to the root of the first sacral nerve. Extradural tumors of this type attached to sacral roots are rare; they are more frequent in the upper segments. The removal of a small tumor of this sort is considered by the author as a distinct advance in the diagnosis of such lesions. The case also proves the need of doing a complete laminectomy, rather than the less radical procedures, with which such small tumors might be missed.

The second case was that of a 55 year old man who gave a history of recurrent left sacroiliac and sciatic pain of thirteen years' duration. For two years the pain was more intense and continuous, becoming definitely worse with coughing and sneezing. For a year he complained of paresthesias in the left foot, calf and thigh. Examination showed spasm of the paravertebral lumbar muscles, atrophy of the left leg and thigh, absence of the left ankle jerk and positive Neri and Laségue signs. Sensory changes were found on the outer aspect of the leg and thigh, with a band of anesthesia in the outer aspect of the left foot. A roentgenogram showed six lumbar vertebrae. The spinal fluid was normal. Injection of contrast medium showed two indentations in the column, which fused into one of the lateral and oblique plates. Laminectomy revealed an irregular, multilobulated tumor, 40 by 32 by 28 mm., which proved to be a neurinoma. The tumor arose from one of the lower lumbar nerve roots and extended outside the intraspinal canal. The author emphasizes the necessity of exhausting all the procedures in the diagnostic armamentarium when analyzing refractory sciatica. The remittent course in the 2 cases reported is interesting.

N. Savitsky, New York.

Peripheral and Cranial Nerves

Meniere's Famous Autopsy and Its Interpretation. Miles Atkinson, Arch. Otolaryng. **42**:186 (Sept.) 1945.

Misinterpretation of Ménière's description of his famous autopsy has led to widespread belief that the disease is due to hemorrhage into the labyrinth. Ménière read a paper with the account of the autopsy in 1861, at which time he referred to an earlier account of it. The two accounts differ slightly, but both describe a "plastic, reddish lymph . . . an exudation" in the labyrinth. In no instance did Ménière speak of blood in the labyrinth. Atkinson urges that since hemorrhage into the labyrinth is not the cause of Ménière's disease a widespread effort should be made by lecturers and writers to expunge this false doctrine, for it impedes understanding.

Ryan, Philadelphia.

Herpes Zoster Auris ("Geniculate" Ganglionitis). Hugh O'Neill, Arch. Otolaryng. **42**:309 (Nov.-Dec.) 1945.

Aural herpes zoster, with or without facial paralysis, is not a rare disease but is constantly misdiagnosed as eczema, Bell's palsy, "barber's itch" and other variants. That the geniculate ganglion is necessarily involved in herpes zoster auris is an unproved assumption. Since the appearance of Hunt's original papers, the case

for geniculate ganglionitis has rested on the lack of necropsy material, the difficulty of disproving his assertions without such material and the variable innervation of the external ear by the fifth, seventh, ninth, tenth, second cervical and sometimes the third cervical ganglia. That the geniculate ganglion may also be involved cannot be denied but that such involvement constantly produces otalgia and aural herpes zoster, as Hunt asserted, is not in accordance with observed clinical data. O'Neill states that the "entity which Hunt labored so strenuously to establish has received in the past forty years a minimum of pathological support." He has overemphasized the dermatologic aspects of the disease, while neglecting the neurologic history. It would seem that until such time as necropsy material substantiates more completely Hunt's assumptions that the geniculate ganglion sensorily innervates not only Hunt's primary cone, the concha, tragus and the auditory canal, but also at times the postaural region, the nares and palate, herpetic involvements of these alleged regressive areas of the seventh nerve should not be referred to as "Hunt's syndrome."

O'Neill reports 8 cases, 1 that of a child of 6 months. In all these cases the variable nature of the disease at its onset and during its course is evident. The fallacy of assigning all cases displaying herpetic aural lesions, usually representing multiple cranial sensory involvement, primarily and solely to the geniculate ganglion because in some cases of aural herpes the triad of otalgia, facial paralysis and aural herpes manifests itself, is apparent.

RYAN O'NEILL, Philadelphia.

PROGNOSIS OF THE RECURRENT NERVE PARALYSIS FOLLOWING THYROIDECTOMY. ALEXANDER F. LASZLO and CHARLES O. FIERTZ, Arch. Otolaryng. **42**:372 (Nov.-Dec.) 1945.

Injury to the recurrent laryngeal nerve with its sequelae in the larynx is one of the unpleasant complications of thyroidectomy. Loss of voice or hoarseness often causes more concern, but the possibility of suffocation in times of stress, due to a limited breathing space, should not be overlooked.

In determination of the possibility of recovery, teamwork between the laryngologist and the neurologist is paramount. The laryngologist must furnish an accurate diagnosis. A technic is described for applying both faradic and galvanic currents while the laryngologist observes the response in the cords and laryngeal musculature: no movement in the paralyzed cord, movement in the arytenoids or in the cord itself. If a faradic response is obtained, prognosis is good for recovery in two months. If, in the absence of any faradic response, the cathodal closing contraction is greater than the anodal closing contraction, the second stage of the reaction of degeneration is present, and recovery will require nine months. A reversal of the formula means a complete reaction of degeneration, and the outlook is poor. Complete severeance of a nerve cannot be exactly determined, but if after two months of treatment there is no improvement, continuation is useless. However, while there is still any sign of life in the innervation of a cord, improvement may take place after eighteen to twenty-four months of electrotherapy. Having used the procedure for at least two years, in hundreds of cases, the authors feel sure it can be a definite diagnostic and prognostic aid, helping those who have experienced phonic disturbance or loss after operation on the thyroid gland.

RYAN, Philadelphia.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Irving H. Pardee, M.D., *President, in the Chair*
Regular Meeting, March 4, 1947

Successful Treatment of a Eunuchoid Man. Dr. George H. Hyslop.

When first seen in January 1943, the patient was 23, married and a divinity student. He sought help primarily because of his high-pitched voice, which he felt would embarrass him in delivering sermons.

At the age of 12 he was above average height; when he was 16, his voice had not changed, and there was excessive flesh on the abdomen and hips. On his body the only hair was a small amount over the pubis, and no hair had developed on his face. Although the external genitalia were extremely small, he had some sexual desire. He married at the age of 20. Erection and orgasm sensation occurred, but there was no seminal discharge. Because the patient regarded himself as incapable of fatherhood, he and his wife had adopted an infant.

The paternal ancestors were normal in height and weight. The maternal grandmother was less than 5 feet (152 cm.) in height, and the maternal grandfather was a little over 6 feet (182 cm.) tall and weighed 200 pounds (90.7 Kg). There was no thyroid disease or diabetes.

The patient was an only child. As an infant he fell from a swing and broke his nose, and at the age of 9 he was struck in the right eye by a baseball, with resulting loss of vision.

Examination showed the characteristic eunuchoid habitus, with long, large legs, a small trunk and feminine distribution of flesh. The skin of the face was delicate and of smooth texture, with not even a prepubertal fuzz of hair. The testes were about the size of lima beans, and the penis was 1 inch (2.54 cm.) long. The voice was high pitched.

Gonadotropic substance by injection twice a week supplemented with 10 mg. of methyl testosterone twice a day was prescribed. By May 1 the patient had noted spontaneous erections, improvement of physical sexual sensation in intercourse and the appearance of seminal discharge. He had lost flesh about the hips and the lower part of the abdomen. There was no change in his voice. The testes were larger and firmer. There was slight appearance of hair over the posterior arch of the zygomas.

Administration of gonadotropic substance was discontinued, but the dose of methyl testosterone was maintained. By August 1 the patient's voice had reached the tenor register. Sexual function had further improved. The skin of the face was becoming coarse, and there was definite hair on the upper lip.

Administration of methyl testosterone was continued, and by November 8 the penis had increased in size and the patient's voice was almost in the base register.

Early in December the patient had jaundice, without pain or temperature, but his appetite was poor. Because the jaundice persisted, the patient was studied thoroughly. In complete roentgenographic and biochemical examinations it was found that the hepatic and renal functions were moderately impaired. At this

time the possibility of idiosyncrasy to the 17-ketosteroid as a cause of the jaundice was seriously considered. At the Presbyterian Hospital a number of patients receiving this drug had had jaundice.

The patient had been doing work in a warehouse over a wharf, with the possibility of contact with rats infected with spirochetal jaundice. Laboratory measures directed to the detection of this possible cause of the jaundice gave negative evidence.

The methyl testosterone was discontinued about Feb. 1, 1944. By March 15 power of erection had decreased; the hair on the face was fading, and the patient's voice was rising in pitch.

The patient was allowed to resume the use of methyl testosterone and was instructed to have routine examinations of his urine once a week. He left town and was next heard from on Nov. 28, 1944. He had continued using the methyl testosterone, with beneficial effects and without any evidence of jaundice or urinary symptoms. He suspected a recurrence of jaundice early in 1945, but apparently this symptom did not actually develop. He continued medication and was studied at the Mayo Clinic and by a physician in Cincinnati. Tests showed that there was residual damage to the liver, and the patient was placed on a low fat diet and was advised to have a hepatic function test every two months while he continued to take the methyl testosterone.

The patient next wrote on Dec. 11, 1946 to report that an 8 pound 6 ounce (3,798 Gm.) daughter had been born to his wife on Nov. 1, 1946. He said that he was "getting along fine" and was using 30 mg. of methyl testosterone a day.

The case is presented for two reasons: (1) the unusually successful result of administration of testosterone to an adult eunuchoid patient, and (2) the question whether this drug may cause at least transient hepatic and renal damage. The literature available to the author contained no reference to such intoxication. On the contrary, the drug has been found to increase renal function.

The possibility that this patient's jaundice may have been due to spirochetal infection has not been conclusively excluded. Such an infection may result in later recurrence, with reappearance of jaundice. Further study of patients at Presbyterian Hospital who had jaundice during the administration of methyl testosterone led to the conclusion that the factor responsible was not the drug but an infection.

DISCUSSION

DR. IRVING PARDEE: I should like to show several slides of a similar case which may be of interest.

The patient was first seen at Mount Sinai Hospital, at the age of 17, and was operated on by Dr. Charles Elsberg for a craniopharyngioma. The operation was successful, and the patient's condition followed a more or less uneventful course up to the age of 29, when he was admitted to the New York Neurological Institute (slide). He was undersized, being under 5 feet in height, and had somewhat gerodermic facies, with puffiness and a slightly yellowish appearance of the skin. He was quite hairless, and his sexual development was that of a newborn infant; one testis was not descended at all, and there was a small nubbin of testis on the left side.

The roentgenogram showed a large sella turcica and operative defect. Neurologically, there was nothing that could or should be done at this time, as he presented no symptoms and there was no increase in intracranial pressure. However, he was much concerned over his feminine voice and his limited sexual

development; and we found on examination that he had a low basal metabolic rate, about —25 per cent. In these circumstances, there was administered thyroid therapy, which made a striking improvement in his general well-being and appearance. In the hope of creating a better psychologic attitude, and perhaps of helping the patient to feel like a new man, as it were, I gave him a large amount of testosterone propionate over a six month period. The results, from his standpoint, were very satisfactory in that there was a definite change in the facial appearance, the areolas became somewhat enlarged, the skin looked much healthier and there developed a fairly good-sized phallus, which was capable of erection and penetration, as well as a moderate, though not complete, growth of pubic hair. The left testis was still present but small; the right one, of course, was not changed in any way by the medication. But the patient was particularly delighted that when he now talked on the telephone he was called "Mister," instead of "Miss." This made a great difference in his working capacity. I cannot say he went as far as did Dr. Hyslop's patient, who married and produced a child; but he made an improvement which was worth while.

As to the use of testosterone, this drug, as well as many similar synthetic preparations, is potentially a dangerous substance and should not be used without careful consideration of the needs of the patient. In Dr. Hyslop's case the toxic results of taking testosterone by mouth were serious, and similar results are seen with substances like stilbestrol, the synthetic estrogen. Because most of these synthetic products have some potential danger in them, they should be used thoughtfully and not too optimistically. The majority of patients who are given testosterone will obtain maximum results within four to six months. After that, there is not much use in pursuing the therapy. I had a patient, a student at Yale, who had been taking 40 mg. of methyl testosterone daily for eighteen months; he was extremely tense, overactive and keyed up mentally, physically and sexually. He had obtained adequate results from the testosterone treatment, and there was no reason for giving him any more. Its use was first cut down to three times a week and then once a week, and of late he has not been taking any and is still as capable sexually as before. But because he has no spermatozoa, he is going to try a gonadotropic stimulant in the hope of bringing about their maturation. It was hard to make him give up testosterone because he thought it was doing him a great deal of good.

Dr. Hyslop has presented an interesting and instructive case, and one wonders what the patient would have done without any medication. Theoretically, he would not have done anything. Practically, there are persons who do not reach puberty until the middle twenties; they are rare, but one does see them; however, there is no doubt the testosterone brought about an adequate development in his case.

The Arterial Route: A Study of Heredodegenerative Diseases. DR. OTTO MARBURG.‡

One may distinguish three groups of heredodegenerative diseases: the hereditary scleroses, the lipidoses and the chronic progressive parenchymatous degenerations. Diseases of the last group have a similar pathologic picture: shrunken, lipodystrophic ganglion cells; atrophic nerve fibers; increased cellularity of the glia; less fibrillar glia, but no signs of an acute process and no compound granular cells.

From a comparison of the sites of these processes with the site of the process in well known diseases with a similar clinical picture (comparative pathology), it could be stated that Werdnig-Hoffmann disease (hereditary familial spinal muscu-

‡Dr. Marburg died in 1948.

lar atrophy) spreads via the ventral spinal arterial trunk, as does poliomyelitis and amyotrophic lateral sclerosis. In Friedreich's disease (hereditary spinal sclerosis) the medial developmental zone, the area supplied by the main branch of the dorsal spinal arterial trunk, is primarily affected, as in the senile myeloses. In the lateral funiculus the focus is cone shaped, as in arterial lesions. Marie's disease (hereditary cerebellar sclerosis) in its degenerative form spreads within the cerebellum only into the dorsal half, in the area supplied by the superior cerebellar artery, the ventral half and the dentate nucleus being supplied by three main arteries. The same area is affected in cases of *ataxie cerebelleuse tardive,* the senile equivalent of Marie's disease. But the superior cerebellar artery also supplies the medullary center of the cerebellum as well as part of the pons, this distribution explaining involvement of this region in Marie's disease. Since, however, there is also a secondary degeneration in the pons associated with all cerebellar degenerations, as there is in the inferior olive, the simultaneous involvement of the three structures mentioned is obvious. There is no need to construct a specific morbid entity—olivopontocerebellar atrophy—since in all cases of true Marie's disease simultaneous lesions of the three structures occur. The cortical area affected in Huntington's chorea coincides with the area supplied by the medial cerebral artery, which also supplies the bulk of the striopallidum in the majority of the cases. This explains the site of the process, which is similar to that in senile chorea.

Thus, one may conclude that the noxa in this group of heredodegenerative diseases spreads via the arteries into the areas best supplied with blood.

Werdnig-Hoffmann disease occurs in earliest childhood, Friedreich's disease in later childhood, Marie's disease in adult life and Huntington's chorea in the forties. The most striking change associated with these various ages is the water content of the tissue, and that, in addition to the pathologic process resembling senile alterations, points to the liver, which maintains the water balance in the brain, and to the endocrine glands, changes in which accompany the senium.

DISCUSSION

Dr. Lewis Stevenson: I am sorry I cannot say much on this subject, but it is my belief that certain structures in the nervous system are selectively affected by certain toxins. I do not think one can escape the connection between carbon monoxide poisoning and the degeneration in the globus pallidus. One cannot escape the connection between methyl alcohol and lesions in the optic nerve, and I could mention many curious and repeatedly observed associations between one toxin and a certain structure in the brain. Although it is true that these degenerations which Dr. Marburg has pointed out follow arterial or arteriolar trees, there is increasing evidence that certain structures are affected by certain toxins in a selective way, no matter what the blood supply may be. There is a curious thing about the degeneration in carbon monoxide poisoning, for example. One may say that this is simply a question of the difference in blood supply between the globus pallidus and other parts of the basal ganglia. My experience has been that the commonest site of a vascular lesion in the brain in cases of arteriosclerosis is in the putamen—certainly not the globus pallidus. I feel, therefore, that the destruction of the globus pallidus in carbon monoxide poisoning cannot be due to the vascular supply. It may be caused by a selective action of the carbon monoxide on the structure itself.

Dr. Marburg has presented an interesting idea.

Dr. Abraham M. Rabiner: Dr. Marburg has made out a good case for the development of these disorders on the basis of the blood supply. One wonders,

however, whether there is not too much coincidence. There are blood vessels elsewhere in the nervous system with similar distribution. Why are not these areas involved? Similarly, why should there be such wide variation in the character, degree and extent of the involvement? For example, one sees children affected with one disease; other diseases affect the middle aged. The olivopontocerebellar atrophy of Thomas and Dejerine occurs at the age of 55 or 60. I had a patient who came to me at the age of 60 with a cerebellar disorder. He told me of an older brother in whom a similar picture had developed at the same age. Several years later he came to me with a younger brother in whom a similar picture developed at the age of 60. Is one justified in thinking of a vascular disturbance, and is it logical, in the case of a patient of 60, to invoke the concept of a noxa or toxin entering the nervous system by the blood vessels? Is it not more logical to assume that certain parts of the nervous system, whether they be cells or axis-cylinders, are at birth endowed with only a certain amount of normal activity, and when their time limit expires they run down? That would seem to me to be a more logical explanation of these abiotrophies, or so-called heredodegenerative disorders.

DR. RICHARD BRICKNER: I do not know that I should follow Dr. Rabiner's logic exactly. It seems to me that if Dr. Marburg showed, and I think he did, general anatomic evidence that lesions in these situations do follow the courses of certain blood vessels, one must heed that point. That is the nucleus of his argument.

Dr. Stevenson's concepts and Dr. Marburg's do not necessarily conflict. Dr. Marburg stressed that when lesions exist they follow the distribution of certain vessels. This may apply even to the toxins Dr. Stevenson spoke of, which can affect only certain parts of the nervous system, and not others.

DR. E. D. FRIEDMAN: May I say a word or two in praise of my revered teacher, at whose feet I sat way back in 1908? He still presents the dynamism, familiarity with authorities and sturdy activity in the field of neuropathology which he exhibited years ago. I pay humble obeisance to him for his effort tonight.

I do not think there is any doubt, as Dr. Stevenson has said, that noxious factors are important in the genesis of neural lesions. May I ask you to notice the title, "A Study on Heredodegenerative Diseases"? The so-called thymicolymphatic constitution, which Neusser, Norris, Osler and others spoke of a generation ago, was accompanied with hypoplasia of the whole arterial tree, including a narrow aorta and defective blood vessels at the base of the brain. There is no reason that if the general arterial tree is defective, the vessels which supply the nervous system should not degenerate prematurely, and thus give rise to ischemic lesions.

It is interesting to see in Dr. Marburg's slides how the lesions follow the distribution of certain vessels. In the early work on subacute combined degeneration it was pointed out that the hypothetic toxin which leads to spinal degeneration gives rise in the early stage to wedge-shaped areas of demyelination, which then fuse into areas of system degeneration in the cord. These hypothetic toxins seem to involve the distribution of the posterior spinal arteries, which supply the posterior columns and portions of the pyramidal tracts which lie close to them.

I feel, therefore, that Dr. Marburg's thinking has a plausible physiologic background. With the addition of a hypothetic noxious factor and the presence of an abiotrophy in the sense of Gowers, lesions would be produced in the nervous system along the lines indicated by the speaker of the evening. We all owe him a debt of gratitude for this suggestive paper.

DR. CHARLES DAVISON: I should like to ask one question. I think that Dr. Marburg spoke of a case of olivopontocerebellar atrophy and mentioned that the lesions were along the distribution of the superior cerebellar artery. If this is true,

why does one see involvement of the superior cerebellar peduncle, which is supplied by the superior cerebellar artery, in cases of olivopontocerebellar atrophy? As is well known, the peduncle involved in this disorder is the middle cerebellar peduncle.

DR. H. A. RILEY: It gives me great pleasure to congratulate my old friend and colleague on his presentation, and I am glad that there were so many people here to hear him. We can all profit by listening to anything that Dr. Marburg has to say.

The one thing that disturbs me about Dr. Marburg's thesis is that he apparently does not find any trouble with the vascular tree. In fact, he blames the very good condition of the vascular tree for the changes which he describes, and he states that these alterations seem to appear in the distribution of vessels which are the largest and supply the widest area. One would have to conclude, therefore, that there must be some effective noxious agent circulating in the blood which, by reason of excessive circulation, and therefore concentration, is capable of producing these lesions. This seems a difficult thing to accept unless one can identify something circulating in the blood which can be proved to be deleterious and capable of bringing about a disintegrating influence in the territory accidentally supplied by the contaminated blood.

DR. OTTO MARBURG: It is self evident that in the limited time of twenty minutes I cannot tell you all of what I have found in many years of study. I shall try to answer the questions asked.

First, it was objected that there is not always a strict distinction between putamen and globus pallidus. This is possible. I have seen cases in which the pallidum was nearly intact, but that depends on vascularization. The vascularization is so variable that one cannot tell from one case to another what will happen.

Second, age is a disease. When I speak of age, I mean pathologic age, and in pathologic age there are changes—in the endocrine glands, as well as in other organs. It is not so far fetched when I speak of the liver, for in Wilson's disease the liver is affected and the water metabolism of the brain depending on the liver is much disturbed. A short time ago, in a case of cirrhosis of the liver I observed changes in the brain not to be explained in any other way than as a disturbance in water metabolism.

I cannot answer all the questions, for it would take too much time and I want Dr. Davison to speak about his interesting cases; but I have written a book, which I hope will be printed, and you will find the answers to all you ask in this book.

Fatal Cryptogenic Neuropathy. DR. SAMUEL BROCK and DR. CHARLES DAVISON.

This paper was published in a previous issue of the ARCHIVES (**58**:550 [Nov.] 1947).

DISCUSSION

DR. H. HOUSTON MERRITT: It is a pleasure to hear Dr. Davison present this paper so clearly and concisely. It is hoped that soon there will be no need for the presentation of cases such as these because we shall understand the causes of all forms of polyneuritis. Recognition of the various causes of polyneuritis is progressively increasing, and it is becoming rare to encounter a case in which the cause cannot be assigned. It has been learned, for instance, that periarteritis nodosa may be a cause of polyneuritis and that it may be a fleeting phenomenon. This vascular condition may be recognized at biopsy, but by the time the patient dies it may have disappeared. It is now recognized that amyloid disease, metabolic disturbances and many other processes may be causes of polyneuritis. Un-

fortunately, there is no good explanation of the neuropathy in any of the cases presented by the authors except in the last, which might be included in the group of neuritides which accompany blood dyscrasias. In that case the blood dyscrasia was confined to the reticuloendothelial system.

There is a interesting series of cases which occurred fifteen years ago, mostly in Cincinnati, but also in other parts of the country, in which people drank Jamaica ginger which was contaminated with triorthocresyl phosphate. The disease at the time of onset looked like a simple polyneuritis; but as time went on it became evident that the spinal cord was affected, and many years later, at autopsy, it was shown that there were diffuse changes in the blood vessels of the spinal cord. The degenerative lesions were secondary to the changes in the blood vessels.

I am glad that the authors did not emphasize too much the diagnostic significance of the albuminocytologic dissociation in the spinal fluid. Too much has been made of this in the literature. Every patient who has a high protein content of the spinal fluid is said to have the Guillain-Barré syndrome. The protein level of the spinal fluid is high in a great many types of polyneuritis. The cause of infectious polyneuritis, or the so-called Guillain-Barré syndrome, is not known. Some think it is caused by virus. A virus has never been isolated, although adequate studies have been made. None of the cases presented by Dr. Brock and Dr. Davison fits into the syndrome which is classic for the so-called infectious polyneuritis.

Dr. H. A. Riley: As is often the case, the discussion hinges on the use of words. I do not see on what basis this condition is described as a polyneuritis. Although entitled a neuropathy, the condition was discussed by Dr. Davison as a polyneuritis. The term neuropathy implies involvement of the nerve distal to the dorsal root ganglion, and not proximal to it; from that point proximally, the root begins, and a disease process which affects it should be called a radiculitis or a radiculopathy. From the sections shown by Dr. Davison of the lumbar portion of the cord, surrounded as it was by nerve roots, it was evident that every one of these structures was almost completely degenerated. These roots must have belonged to the cauda equina between the point of exit from the central axis and the ganglia situated in the intervertebral foramens. The condition is certainly a radiculopathy at least, and the evidence in the sections indicates that it is also an encephalomyelopathy. Abbott and Evans described a series of patients who presented evidence of an acute degenerative encephalomyelopathy and radiculopathy, the porphyrins being responsible for the condition (*Bull. Los Angeles Neurol. Soc.* **11**:20-31 [March-June 1946]. In his discussion of the third case, Dr. Davison noted that no porphyrins were found in the urine; I wonder whether it is possible that in any of his other cases there could have been porphyrinuria. Did acute gastrointestinal symptoms precede the onset in any of the cases?

Dr. Charles Davison: Only 1 of our cases was studied for porphyrinuria, and none was found. Gastrointestinal symptoms were not observed in any of the cases.

Dr. H. A. Riley: My further remarks have nothing to do with the presentation, but it is extremely interesting what extensive involvement these patients may have and yet not always die. At the Institute, my colleagues and I saw a patient, a girl of 18 or 19, who had a typical virus encephalomyeloradiculopathy, with albuminocytologic dissociation and a characteristic history of acute progressive disease. There was evidence of involvement of the roots and central nervous system. The process was so extensive that practically all the distal muscles were paralyzed by the process, and her condition resembled advanced amyotrophic lateral sclerosis. Her musculature became so affected that she had to be put in a respirator. Her character became radically different from that which she had shown

before the illness, and she was crabby, irritable and restless. This condition continued for a month or six weeks, and then she began to improve; her muscles resumed their usual contours, and she left the hospital. She returned to us a month after she was discharged, and one would not have known that anything had ever been the matter with her. She was dancing and doing anything she wished to do. She had been completely paralyzed peripherally and almost completely paralyzed centrally. It is possible, therefore, for this condition to go almost to a lethal termination and the patient then recover.

DR. H. H. MERRITT: Did she have porphyrinuria?

DR. H. A. RILEY: I do not think we looked for it. I believe her case was one of virus encephalomyelitis.

DR. SAMUEL BROCK: I sympathize with Dr. Riley's interest in nomenclature; he and I have spent many hours together discussing semantics in connection with neurologic terminology. It is true that the term "neuropathy" is not all-inclusive, but the point we meant to make was that the main impact of the pathologic process fell on the peripheral nervous system, including the roots. If one wishes to appease one's nomenclatural conscience, we can speak of neuropathy and radiculopathy; indeed, one might go up the entire axis, adding various prefixes to "pathy."

We were also interested in the question of albuminocytologic dissociation. What is the neuropathologic basis of this dissociation in the course of these inflammatory and associated reactions? It is a general feeling that as the acute disease passes off and the patient either recovers entirely or is left with a few residua all the constituents of the spinal fluid return to normal. This is not always so. In 1939 a patient (slide) became ill with what looked clinically a bit like multiple sclerosis or acute disseminated myeloradiculoneuropathy. In the spring of 1939 the spinal fluid protein varied from 196 to 392 mg. per hundred cubic centimeters. It is interesting to note that after this acute flare-up he recovered, but not entirely, and has remained in status quo for five or six years. He has returned to the Neurological Institute for many subsequent lumbar punctures. Now, for seven years, this patient's spinal fluid protein has never gone below 200 mg. per hundred cubic centimeters; 176 mg. was the lowest of the seventeen estimations and 514 mg. the highest. The last examination of the fluid was made in February 1946. This man has not been acutely ill for six years, and yet his spinal fluid shows increased protein, every other constituent being normal. There must be other instances of this persistent increase in the protein content of the spinal fluid among cases of so-called cured, or stationary, neuropathy; but it would be going too far afield to attempt any explanation of this phenomenon now.

DR RICHARD BRICKNER: Dr. Riley may remember several patients we saw several years ago, in prewar days, at the Neurological Institute. They had meningomyelitis, with a high degree of pleocytosis. They made fairly rapid clinical recoveries. We meant to keep them in bed until the cell count of the spinal fluid returned to normal. Long after they were otherwise well these patients retained their pleocytosis, and they did so until we finally let them up, and even discharged them from the hospital. The duration of observation was not longer than about six weeks.

DR. E. D. FRIEDMAN: In a case of which Dr. Davison spoke the patient showed changes in the fundus.

DR. CHARLES DAVISON: Yes, and pathologically there was perivascular demyelination in the optic nerves, tracts and chiasm.

DR. E. D. FRIEDMAN: What reason was there for not considering the possibility of a neuromyelitis optica with polyneuritic manifestations?

DR. IRVING PARDEE: In connection with Dr. Brickner's discussion, I should like to mention 2 patients whom we had under observation for several years after recovery from a typical Guillain-Barré syndrome. They continued to have a high protein content of the spinal fluid, 250 or 300 mg. per hundred cubic centimeters, for several years after they had made a good clinical recovery.

DR. H. A. RILEY: If you are thinking of the same patient that I am, the man has never been well since; he has continued to have some cerebellar signs.

DR. SAMUEL BROCK: He is not well, it is true.

DR. CHARLES DAVISON: Dr. Riley has brought up some interesting points. For a number of years I emphasized that peripheral neuritis, or peripheral neuropathy, is really not true peripheral neuritis or neuropathy, for frequently other parts of the central nervous system are also involved. This was one of the reasons for tonight's presentation. These cases were studied by careful observers, all of whom agreed in calling the condition peripheral neuritis. There was no question that patients presented a clinical picture of peripheral neuritis with some involvement of the cranial nerves. Pathologically, other parts of the nervous system were involved. I have seen many instances of peripheral neuritis without clinical evidence of lesions in other parts of the neuraxis. At autopsy lesions were observed in the roots and in the posterior columns.

As to the terminology, Dr. Riley may be right that neuropathy refers to the entire central nervous system. Perhaps we should have used the term "peripheral neuropathy." We have to make some distinction between the infectious and the noninfectious type. The latter should be called a neuropathy. As he is more proficient in nomenclature than I, I am awaiting his decision.

As to Dr. Friedman's question, the optic nerves, chiasm and tract which I showed in the first case, disclosed tiny perivascular areas of demyelination. I am familiar with most of the cases of so-called neuromyelitis optica reported, and in those cases the chiasm and the tract showed extensive areas of demyelination. Personally, I know of no cases of neuromyelitis optica with involvement of the peripheral nerves. Furthermore, in neuromyelitis optica the spinal cord shows extensive lesions more like those seen in multiple sclerosis. As a matter of fact, neuromyelitis optica is only an acute form of multiple sclerosis. As to changes in the fundus, Dr. Merritt will corroborate the statement that in many instances of peripheral neuritis some changes occur in the fundus. In 2 of the cases I presented such changes were present.

NEW YORK NEUROLOGICAL SOCIETY AND NEW YORK ACADEMY OF MEDICINE SECTION OF NEUROLOGY AND PSYCHIATRY

George Baehr, M.D., *President, New York Academy of Medicine, in the Chair*
Combined Meeting, Feb. 6, 1947

Intrinsic Asthma. DR. FRANCIS M. RACKEMANN (by invitation), Boston.

In the beginning all was allergy that wheezed, and if the methods peculiar to allergy could not reveal the cause these methods were deemed faulty. It was recognized, however, that secondary infections could aggravate the simple allergic process and then that primary infections could produce a "bacterial allergy." More recently chronic focal infections, such as those of the teeth and sinuses, have been recognized as sufficient to explain chronic asthma. It is quite evident that all is not allergy that wheezes.

Whether one should consider "asthma" as a disease with a basic mechanism common to all cases, or whether one should continue to regard the wheeze as

merely a symptom exhibited by a variety of different processes is an important question not yet answered.

The study of the classification of patients with asthma reveals a variety of types. The young woman who is sensitive to cats, or who wheezes when her ragweed hay fever becomes severe, presents a very different picture from that shown by the older "tired business man" or by the "harassed housewife." The young lady is allergic; she is sensitive, but the exciting cause of trouble is outside her body. The older patients carry the cause of their trouble with them as an "intrinsic" disturbance, which may be either psychogenic or somatic, and is often both. A study of the "cures" which should be profitable, fails to show any common factor which explains the "cures." Such a study, however, shows an important point—that in certain instances the patient who has been free from asthma for ten years or more has a relapse. Evidently, there is a disturbance which is fundamental and which persists—a "weak spot" which flares under provocation of all sorts, with allergy one of the precipitating factors. In this sense it may be wiser to regard asthma as a disease, and not as a mere symptom. More attention must be directed to that part of the mechanism which is basic and fundamental.

The problem is still open; the approach to its solution is not at all clear.

This paper was published in the *Bulletin of the New York Academy of Medicine* (**23**:302-306 [May] 1947).

Psychosomatic Aspects of Allergic Disordes. Dr. Edward Weiss, Philadelphia (by invitation).

The allergic and the neurotic population are so large that they must overlap. If, for no other reason, therefore, allergic and neurotic disorders will exist in the same patient. However, personality studies suggest a more intimate connection—a specific relation between neurotic character structure and allergic disorder—possibly representing manifestations of the same basic fault, the one discharging on the level of psychic representation, through thoughts and feelings, and the other on the physiologic level, by means of disturbances in organ function.

Psychosomatic study of an allergic problem, therefore, utilizes separate technics —psychologic and physiologic—applied simultaneously; and diagnosis must be established not simply by exclusion or evaluation of physical factors but with additional positive evidence of personality disorder meeting certain psychosomatic postulates. This will demonstrate that in a given case physical and psychologic factors act in a complementary fashion to produce the disorder—in one case specific physical factors, and in another specific emotional factors, may predominate. The latter seem to be determined by certain trends within the personality; just as oral attachments seem to determine gastrointestinal disorders, so respiratory and dermal attachments (to the mother) apparently determine respiratory and dermal allergic manifestations (Saul).

Migraine seems more closely related to the character structure and personality trends observed in patients with essential hypertension, having to do with the amount and disposition of hostile impulses.

The allergic disorders seem to fall for the most part into the group of organ neuroses that can be termed vegetative (Alexander), representing early and profound deviations of personality development. What role the constitution may play cannot be determined—no methods are available to delimit constitutional and acquired factors. One can, however, evaluate physical and psychologic factors, and proper management depends on such evaluation. Then psychotherapy together with the allergic approach will mean better treatment for the patient with an allergic disorder.

Book Reviews

The Human Race. By Emil Froeschels. Price $3. Pp. 197. New York: Philosophical Library, 1947.

The author, who is both a physician and a philosopher, extols the freedom of the will and the junction of each and every human being with the infinite. In this study of the nature of knowledge, it is felt that the knowledge of God, of the infinite, of the universe, as well as of the freedom of will, all of them common to every human being, if evaluated properly, should prove that the differences between all human beings are negligible as compared with the treasures they have in common.

This book is divided into three major portions, concerned with the problems of congenital knowledge, two different kinds of time and, finally, a discussion of our destiny as members of the human race.

Part I deals with congenital knowledge of the infinite, the unity and existence of God and the concept of the unconscious. Since the infinite and God are within our psychic person, and are not derived from any knowledge acquired throughout our lifetime, the author concludes that this must, therefore, be congenital knowledge. Of psychiatric interest is his rejection of the right to speak of the unconscious in the human personality. Since we cannot perceive and conceive to know anything about a supposed existence of an unconscious within us, since such a quality would be characterized by perfect unawareness, the author prefers to substitute the terms "expression-ripe" and "non-expression-ripe" for degrees of awareness.

Part II concerns time; the existence of the concept of rest, and motion as such, as recognizable only as the opposite of this concept; being and becoming; the will; reality and truth, and other concepts.

Part III interestingly discusses mistakes, and our ability to know the truth, as due to the difference between the two ways in which the universe offers itself to us, namely, as a whole, on the one hand, and in parts, on the other. Herein, the author feels, lies the permanent source of mistakes. As perception of all parts of the universe is impossible with our limited time and senses, mistakes will exist as long as mankind exists. This is not defeatism, since our congenital knowledge is not limited and is the knowledge of the universe. The author concludes that the hope must always remain that men, who are equal participants in the community of infinite knowledge, may become aware of this community, act in accordance with infinite values and discard the more obvious finite elements, whose essence is division and whose effect may be discord. Thus, the knowledge that the infinite is the end-all and be-all of existence should teach wisdom and bring a clearer realization of the real value.

The reviewer highly recommends this book as interesting, provocative and stimulating in its approach to a new freedom for man.

Freud on War, Sex and Neurosis. Edited by Sander Katz, with preface and glossary by Paul Goodman; translated by Joan Riviere, and others, Price $3. Pp. 288. New York; Arts & Science Press, 1947.

Joan Riviere, and others, Price $3. Pp. 288. New York; Arts & Science Press, 1947.

This is not a commentary on Freud's views, as one would suspect from the title. It is an actual collection of some of Freud's lesser known writings—"lesser known" in the sense that they are generally not known to the layman but are for the most part well known to any serious student of psychology.

The selections are good ones, and they are offered without any commentary or criticism. While this particular collection is meant for the layman, it is not meant for the average reader. I do not believe that Freud addressed himself to the layman when he wrote these papers. The great furore in medical circles that was created by the "Dora case" when it was first published will probably have the same effect on the lay public today. The paper is a masterpiece of clarity, but it will meet with the same psychologic resistance that all psychoanalytic theory encounters. The first paper in this collection, entitled "One of the Difficulties of Psychoanalysis," is, again, a masterpiece of clarity, but, unfortunately, to explain resistance does not eliminate it.

On the other hand, with the deluge of literature on psychology and psychiatry that the postwar period has brought, it should be most gratifying to hear from the master himself. So many misinterpretations of freudian conceptions have been made that it is about time the intelligent reader be given an opportunity to get to the source and judge for himself. Freud's papers on "Sexual Morality and Nervousness," "A Special Type of Object Choice" and "The Most Prevalent Form of Degradation in Erotic Life," all of which appear in this book, are clear expositions of subject matter which is currently receiving a great deal of confused literary treatment. The acceptance of Freud's ideas may not be easy, but at least the reader will know what these ideas are.

The latter statement needs some qualification. It is difficult to imagine a reader plunging into the "Dora case," for example, without a previous knowledge of Freud's "Three Contributions" and his work on "The Dream." Furthermore, Freud's style, though simple and clear, makes every sentence so pregnant with psychologic insight that his papers require a concentrated effort. His eloquence makes one want to sit back and read easily and with relaxation. It can be done, and his papers will be "enjoyed." But to understand the implications and ramifications of each statement will require a more serious effort at concentrated study.

This reviewer does not believe it was the intention of the editors of this book to present the psychoanalytic theory of human behavior. Its purpose was to present Freud as a keen essayist, a master of wit, irony and psychologic insight. Any one who reads these collected papers cannot fail to recognize Freud as the literary genius that he was.

Psychiatrists who have never gone through Freud's collected papers owe it to themselves to read this particular collection. They may not agree with Freud, but they will enjoy these papers.

If A Man Be Mad. By Harold Maine. Pp. 435. New York: Doubleday, Doubleday & Company, Inc., 1947.

Dissatisfaction with public hospital care of the mentally ill has recently shown a healthy tendency to a national increase. The popular interest in psychologic themes and in the problems of mental health and ill health finds a certain practical reflection in the susceptibility of members of the medical profession and the more intelligent members of the laity to an arousal of guilt feelings regarding the generally shabby and inadequate provisions made by the public hospital. Numerous books, magazine articles and even some moving pictures have recently served this function. Members of the medical profession are apt to take refuge in one of two highly justified, but not entirely satisfactory, positions—to say either that "Not being a psychiatrist I have no more to do with this than any other citizen," or "Even though I am a psychiatrist or an interested citizen, the administration of the public hospitals is out of my hands because it never was a 100 per cent *bona fide* medical project anyway, but was a project of social quarantine." Both these posi-

tions are unsatisfactory because progress in psychiatry and hospital administration and in public awareness have all reached the point where one should recognize that the best scientific medical care of the committed psychiatric patient is in the long run the most economical. This implies the obligation of the medical profession to be active in a reformation of present practices.

A comprehensive fact-finding study regarding public psychiatric hospitals would be extremely expensive, and such books as this one serve the valuable purpose of giving one a first hand impressionistic report on which certain tentative hypotheses can be based. The writer is a man of literary talent whose candor, earnestness and sincerity seem indubitable. He makes no attempt to conceal the way in which he became actively involved in reform. He was admitted to state, government and private hospitals repeatedly for addiction to alcohol and in certain respects made himself a nuisance both inside and out. The experiences, however, gradually had a certain cumulative effect; miserable and disordered as was his personal life, the hospital world into which he was thrown seemed worse. He was so shocked at the lack of medical control, the shortage of physicians, the ignorance and cruelty of the ward personnel and the futility of the entire system that he relinquished his own neurotic preoccupations for a dedication to psychiatric hospital reform, with particular reference to the problem of attendants. He obtained employment in several hospitals as an attendant and learned from the inside how unmedical, unscientific and actually inhumane this system can be, and in many places is. Since writing the book, the author has, to the reviewer's knowledge, continued his constructive campaign to improve the status, ideals, education and technics of hospital aides and attendants.

Cerebral Angiography with Perabrodil (Carotid Angiography). By Arne Engeset, M.D. Price, $8. Pp. 207, with 75 illustrations. Oslo, Norway: Fabritius & Sons, 1944.

Perabrodil is an organic iodine compound that can be used safely in the delineation of the cerebral vessels. Angiography has become such an important method of diagnosis of intracranial lesions that every neurologist should have a reasonable acquaintance with this subject. Not infrequently the arteriogram is more valuable than the encephalogram or the ventriculogram, for it reveals not only the location of the disease process but also its nature. In a case of rapidly expanding intracranial lesion, for instance, the arteriogram can distinguish abscess, hematoma and gliobastoma multiforme. Subarachnoid hemorrhage from aneurysm can be differentiated from that due to angiomatous malformations and can guide the surgeon in his surgical attack. By indicating the principal source of blood supply in certain tumors, the angiogram can point the way to satisfactory control of hemorrhage at the time of operation.

Whether the contrast medium is injected into the exposed artery or whether it is done by needle (obviously requiring a modicum of skill), the teamwork of the neurologist and the roentgenologist must be well nigh perfect.

Engeset covers the subject of arteriography in somewhat sketchy fashion, but his illustrations are demonstrative.

Foundations of Neuropsychiatry. By Stanley Cobb, M.D. Fourth edition, revised and enlarged. Price, $2.50. Pp. 260. Baltimore: Williams & Wilkins Company, 1947.

In the latest edition of his widely known volume, Dr. Cobb has enlarged on previous editions, and a good deal of the material on cerebral circulation, the autonomic nervous system and the motor cortex has been rewritten. The basis of

modern neurology is presented in a most gratifying and inclusive, yet succinct, manner. Clear discussions of important and significant anatomic, physiologic and pathologic data are based on classic observations and experiments. Controversial matters are mentioned where indicated, as are the many unsolved problems demanding further investigation. The sections on motor integration and localization, functional localization in the cerebral cortex and consciousness and the problem of the "mind-body" relationship present an epitome of neurologic and neurophysiologic thought "as of 1947." Total behavior patterns are stressed, rather than component ones, and dualistic thinking in regard to "functional" versus "organic" derangements is condemned as being illogical, confusing and arbitrary. Thus, "The body acting as a whole is an organism; anything that happens in that organism is organic. Structure and function are inseparable."

While the orientation is chiefly neurologic in the general sense, the final chapters deal with psychologic concepts in medicine and psychopathology, in which the highlights of these respective topics are briefly summarized.

The work is thoroughly readable and is enthusiastically recommended to all concerned with neuropsychiatry, as well as with general medicine.

The Philosophy of Insanity. Anonymous, with an introduction by Frieda Fromm-Reichmann, M.D. Price, $2.50. Pp. xii, plus 116. New York: Greenberg, Publisher, Inc., 1947.

In the introduction, Dr. Fromm-Reichmann, of the Washington School of Psychiatry, presents this as the autobiography of a "Late Inmate of the Glasgow Royal Asylum for Lunatics at Gartnavel," published anonymously under the title, "The Philosophy of Insanity."

This is an account, in retrospect, of a severe mental illness, hospitalization and recovery occurring in the middle of the nineteenth century. It is a plea for a better understanding and acceptance of the psychotic patient, and for the certain essential similarity of the mentally ill and the mentally well.

The book contains much wisdom, e.g. (page 3), "in insanity as in childhood, the brain is dreadfully susceptible of lasting impressions; and the more absurd, impossible, and unnatural they are, the greater is the difficulty which lies in the way of getting rid of them . . . Delusion may continue long after recovery and is quite compatible with the possession of strength of mind to conceal it. This is what may be called the dregs of insanity and may exist without being allowed to influence the actions."

The book is highly recommended.

Deep Analysis: The Clinical Study of an Individual Case. By Charles Berg, M.D. (Lond.), D.P.M.; Physician to the British Hospital for Functional Nervous and Mental Disorders. Price $3.50. Pp. 254, with a glossary. New York: W. W. Norton & Co., Inc., 1947.

There is no foreword or introduction to indicate whether this work may be of interest to psychoanalysts, psychiatrists or general practitioners. However, the glossary, defining analytic and psychiatric terminology, readily reveals that the book is intended for popular distribution. The publisher's summary on the jacket, on the other hand, states in part:

"Psychiatric literature abounds in case histories and illustrative case material. But for the author of this book there is a lack of satisfaction in such short case descriptions. To remedy this defect he set himself to write the illuminating description of a complete Freudian analysis of a single case . . . . We see and understand the hidden depths of the nature of the human mind, and obtain introductory insight not only into normal mental functioning but into almost all its psychopathic

aberrations, including frigidity, impotence, hate, hysteria, obsessions and even paranoia and schizophrenia—all present in minor degrees in normal personality.

Although of particular interest to the doctor and the psychiatrist, "Deep Analysis" is written in a manner perfectly understandable to the general reader and cannot fail to absorb the attention of any nonprofessional reader who is attracted by its subject."

The book is divided into three parts, entitled "Father," "Mother" and "Son," comprising 23 brief chapters. The first two parts consist in discussions on technic, transference, regression to infancy, father fixation and the homosexual component; mother fixation, heterosexual anxiety and resistance, problem of anxiety, psychology of accidents, analysis and money, self sufficiency of the anal character, transference resistance and the mother image. Part three deals with analysis of the transference and relief of the mother fixation.

Some of the general discussions are sufficiently technical and complex to make this book difficult reading for the laity. Although a glossary is supplied, there is not the clarity of explanation of many of the technical terms and the theoretic psychodynamic consideration which is important for the better understanding of the material by the layman.

Various theories and conclusions expressed by the author will arouse differences of opinion from the standpoint of clinical psychiatry. The various possibilities of clinical diagnosis in the presence of a symptomatic problem are omitted or superficially discussed. Although the author refers to this case study as an abridged version of voluminous material, there is no detailed clinical picture of the life history of the patient, concerning whose personality and personal and family data only a sketchy and unsatisfactory description is given. Perplexity as to the diagnosis is to be noted when one reads on page 18 that the patient's illness is "unclassifiable" and on page 33 is that it is "a form of obsessional neurosis," while on page 157 the author refers to the patient's "delusions," which, however, are not described. Concerning the diagnostic problem, the author argues as follows. "The smug satisfaction we achieve by arriving at a diagnosis may deflect us from our real purpose: that of understanding and curing."

The major portion of the book centers around the patient's fixations to mother and father in infancy and childhood involving incestuous and homosexual inclinations, together with the problem of transference resistance during the analysis. The author employs the free association of thought technic, particularly in the dream interpretations, which are made chiefly by the patient in combination with very active therapy by the analyst. The dialogue between patient and analyst is remarkable in that the patient's conversation contains much self-analytic explanation, so technically worded that one could easily substitute it for the analyst's interpretative discussion. The terminology and the explanation of mechanisms are fundamentally those of basic freudian concepts and theories, without any new or original additions.

For psychiatrists and analysts, the literature contains many examples of extremely detailed deep analyses of excellently presented case material of greater value and interest than that rcorded in this volume.

The Postnatal Development of the Human Cerebral Cortex. By J. Leroy Conel. Volume III: The Cortex of the Three-Month Infant. Price, $12.50. Pp. 158, with 220 illustrations. Cambridge, Mass.: Harvard University Press, 1947.

The present volume represents the continuation of a long term study of the cytoarchitecture of the human cerebral cortex, the first and second parts of which were published in 1939 and 1941. Similar technical methods were used through-

out, permitting comparison of the development of the cortex in the newly born, the 1 month and the 3 month old infant. Development is judged according to histologic criteria, which include number and size of cells, amount of chromaphil substance, degree of myelination and the structure of cellular processes.

The underlying concept is that a correlation may be made between maturation of function and observed structural changes and that this correlation may be made with respect not only to specific areas of the brain but even to layers and individual cells. Point to point correspondence and discrete localization of function in the brain are implicit in this approach. Thus, according to structural criteria, the most advanced area is that representing the upper portions of the trunk, the arm and the forearm, on both motor and sensory sides. This concept is correlated with observations of other investigators that the 3 month old infant shows greatest voluntary activity in the muscles in these regions.

This study should prove of greatest value as a reference work on the normal appearance of the cortex. Approximately half the volume consists of a series of excellent photomicrographs and drawings, while the text contains detailed descriptions of the areas selected for comparative study. Wider investigation will determine how these data are to be used in understanding the maturation of the nervous system.

Man, Weather Sun. By William F. Petersen, M.D. Price, $10. Pp. XIX plus 463, with 294 illustrations. Springfield, Ill.: Charles C Thomas, Publisher, 1948.

Most serious students would not give this book more than the few glances necessary to discover that its main purpose is to demonstrate the following line of argument: Human physiology and psychology are affected by the weather; the weather is affected by sun spots; therefore, economic crises are as inevitable as the periodicity of sun spots; hence, mankind must stop trying to eliminate economic crises and wars and learn to live with them.

However, the investigation of man's relation to daily and seasonal changes in weather has been so neglected that this book has a greater importance than would be indicated by its unreliable methods, its great unevenness in quality of reporting and reliability of observations and its fatalistic, passive approach. It contains many references to earlier investigations, mostly of a statistical nature. There is also included a report of an investigation of a set of triplets by Dr. Petersen over a forty-four hour day period. In this investigation, daily measurements and observations of various clinical and biochemical phenomena were made and attempts made to correlate them with the changes in weather during the same period. Unfortunately, this promising method of investigation became relatively sterile because the author was more interested in proving correlations than in understanding them.

The chief value of this book is that it brings together many of the investigations in the field; the integration of the material is not useful.

Understandable Psychiatry. By Leland E. Hinsie, M.D. Price, $4.50. Pp. 359. New York: The Macmillan Company, 1948.

In this book, as its title implies, Dr. Hinsie attempts to present the basic formulations of psychiatry as to psychodynamic principles, psychopathologic entities and the various disease groups and symptom complexes, in a form which is understandable to both patient and physician. His premise is that "psychiatry cannot succeed as a treatment procedure until the treated person, with the physician's aid, comes to know as much about his or her personality as does the physician."

Dr. Hinsie succeeds very well in presenting his ideas in vivid, yet clear and nontechnical, language. The titles of the chapters themselves ("How Feelings Make

Us Sick," "Emotional Pains," "The Oneness of Mind and Body") indicate his approach, with emphasis throughout on psychosomatic interrelations. There is a liberal use of illustrative case material, woven into the text itself and bringing color and life to the theoretic discussions. The discussions are also full of pungent aphorisms, such as that "people are to the neurotic individual what he conceives them to be."

For his theoretic base, Dr. Hinsie adheres closely to the formulations of classic psychoanalysis. His statement that it is a "prevailing opinion" that there are two basic instincts, the erotic and the destructive, could be challenged, in view of the many opinions to the contrary. His categorical acceptance of a "racial mind" containing patterns "which nature had laid down eons ago" may also surprise many.

In a book of such ambitious scope, omissions and one-sided emphasis are almost inevitable. In this regard, the author's cursory mention of shock therapy is outstanding. On the whole, however, Dr. Hinsie has written a stimulating and informative book, which can be recommended for both the lay public and the physician.

News and Comment

POSTGRADUATE COURSE IN PSYCHIATRY AND NEUROLOGY

A twelve weeks' postgraduate course in psychiatry and neurology will be given by the Division of Psychiatry, University of California Medical School, in cooperation with the University Extension (Medical Extension), University of California, at the Langley Porter Clinic, Aug. 30 through Nov. 19, 1948. This will be a repetition of the course given previously and will be open only to qualified physicians. Instruction will be given under the direction of Dr. Karl M. Bowman, professor of psychiatry, University of California Medical School, with the assistance of staff members from the various divisions of the medical school.

The following subjects will be covered: general psychiatry; child psychiatry; psychobiology; psychoanalysis; psychology and psychopathology; functional and organic psychoses; psychoneuroses; therapy; psychosomatic problems; neuroanatomy; clinical neurology; neuropathology; neurophysiology; electroencephalography; roentgenologic diagnosis, and other related topics.

The fee for the course is $200, payable in advance, by check or money order, to the regents of the University of California. Veterans applying for educational benefits under federal or state statutes are to present certificates of eligibility at the time of registration. The certificate will be accepted in lieu of the fee. Applications should be sent to Dr. Stacy R. Mettier, Head of Postgraduate Instruction, Medical Extension, University of California Medical Center, San Francisco 22. Dr. Mettier will furnish any additional information that may be required.

APPOINTMENTS TO FACULTY OF IOWA STATE UNIVERSITY SCHOOL OF MEDICINE

Dr. Adolph L. Sahs, formerly acting head of the department of neurology, has been appointed head of the department.

Dr. Raymond R. Rembolt is the newly appointed assistant professor of pediatrics, director of the State Services for Crippled Children and medical director of the School for Severely Handicapped Children.

Dr. Wendell P. Stampfli has been appointed assistant professor of radiology.

Dr. Otis S. Lee Jr. has been appointed assistant professor in ophthalmology.

Appointments will go into effect on July 1.

BRONX SOCIETY OF NEUROLOGY

The Bronx Society of Neurology and Psychiatry has recently been incorporated. The officers are: Joshua H. Leiner, M.D., president; Nathan Savitsky, M.D., vice president; Jacob H. Friedman, M.D., secretary; Moses Madonick, M.D., treasurer. The executive committee members are: Hilbert W. Ehrlich, M.D.; Nicholas R. Locascio, M.D., and Joseph D. Sullivan, M.D.

THE ARCHIVES OF NEUROLOGY AND PSYCHIATRY is published by the American Medical Association to stimulate research in the field of diseases and disorders of the nervous system and to disseminate knowledge in this department of medicine.

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CONTENTS

PAGE

Arteriovenous Aneurysms of the Brain: Their Diagnosis and Treatment. HERBERT OLIVECRONA, M.D., AND JOHANNES RHVES, STOCKHOLM, SWEDEN 567

Headache: A Common Symptom of Cervical Disk Lesions; Report of Cases. AIDAN A. RANEY, M.D., AND RUPERT B. RANEY, M.D., LOS ANGELES 603

Sociopsychologic Structure of the SS: Psychiatric Report of the Nurnberg Trials for War Crimes. LEO ALEXANDER, M.D., BOSTON ... 622

Metastatic Tumors of the Central Nervous System: I. Intracerebral Metastases as the Only Evidence of Dissemination of Visceral Cancer. CHARLES RUPP, M.D., PHILADELPHIA 635

Prognostic Significance of Incidental Acute Somatic Disorders in the Course of Psychoses: Reevaluation of Five Cases Fourteen Years Later. ALFRED GORDON, M.D., PHILADELPHIA 646

ABSTRACTS FROM CURRENT LITERATURE 653

SOCIETY TRANSACTIONS:

New York Neurological Society 673

New York Neurological Society and New York Academy of Medicine, Section of Neurology and Psychiatry 681

BOOK REVIEWS 683

NEWS AND COMMENT 690